VOL. LVII, No. 8 AUGUST 1952

VETERINARY MEDICINE

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Highlights of the A.V.M.A. Convention

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ESTABLISHED 1905

PUBLISHED MONTHLY

Entered as second-class matter September 15, 1949, at the post office at Kansas City, Mo., under the Act of March 3, 1879.

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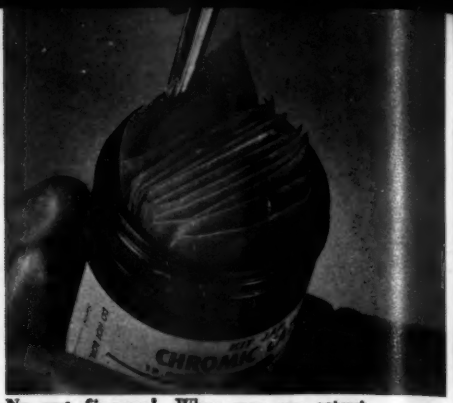

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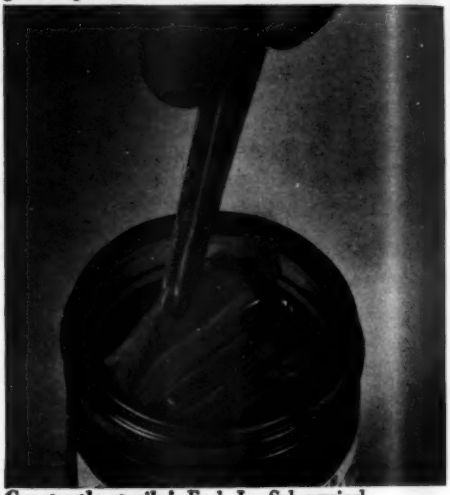

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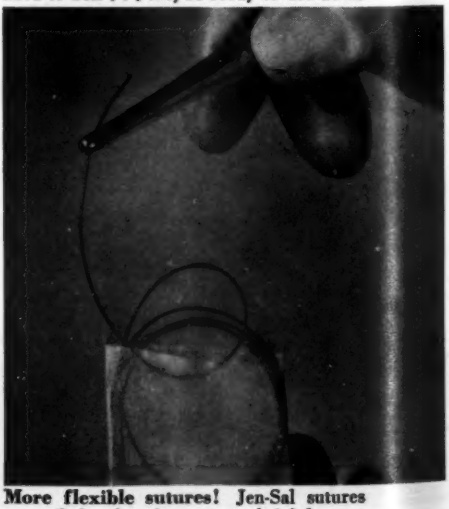

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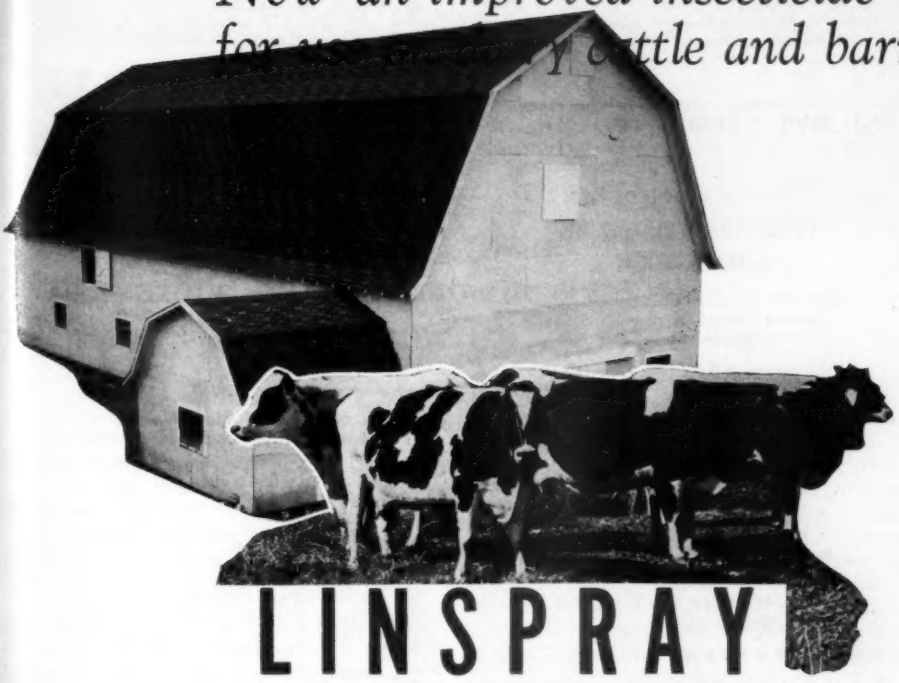

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VETERINARY MEDICINE

A Monthly Magazine Devoted to the Promotion of the Art and Science of Veterinary Medicine

Vol. XLVII AUGUST 1952 No. 8

Published Monthly by the

VETERINARY MAGAZINE CORPORATION

Suite 803, Livestock Exchange Building
Kansas City 15, Missouri, U.S.A.
Telephone: Victor 2985

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SUBSCRIPTION RATES: One year, $5.00; two years, $8.75; three years, $12.00; five years, $18.00. Single copy, 50c. U. S. Funds. Postage prepaid. Authorized agents will present letter of collection authority. Pay no money to others.

Entered as second class matter at the post office at Kansas City, Missouri. Entered as second class matter under the title of the American Journal of Veterinary Medicine, July 19, 1910, at Chicago, Illinois, under the Act of March 3, 1879.

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Nitrofurazone As A Therapeutic Agent In *Salmonella choleraesuis* Enteritis Of Young Pigs

JAMES E. GUTHRIE, D.V.M.,* Ashland, Ohio

THE enteric complex commonly diagnosed in the field as necrotic enteritis is a serious disease of young pigs. It is detrimental to the swine industry from the standpoint of death losses, as well as from reduced efficiency of feed conversion in surviving animals.

That *Salmonella choleraesuis* is one, and probably the primary etiological agent of this condition was demonstrated by Murray, Beister, Purwin and McNutt[1,2] and by Beister, Murray, McNutt, and Purwin[3]. Deficiencies of nicotinic acid and other members of the vitamin B complex have been suggested as contributing factors by Davis, Freeman and Madsen[4] and by Luecke and coworkers[5].

Attempts to reproduce this commonly encountered enteric disorder met with variable results until Schoening, Dale, Mott and Haberman[6] demonstrated that lyophilized virulent cultures of *Sal. choleraesuis* would consistently reproduce the disease when fed to susceptible pigs. Prior to this work it was impossible to undertake therapeutic investigations under controlled conditions because of the variability in virulence of cultures. Slavin[7] also demonstrated that it was possible consistently to infect young pigs with a culture of this organism isolated from an extensive outbreak of paratyphoid and kept dried to maintain uniform virulence. The process by which his culture was dried is not described.

Kernkamp and Roepke[8] conducted a series of controlled experiments on the use of sulfaguanidine in pigs suffering from enteritis contracted under natural conditions. Other workers[9,10] have also conducted clinical investigations on the value of similar poorly absorbed sulfonamides. These studies indicate that this class of chemotherapeutic agents has merit for the treatment of enteric disorders in pigs.

Luecke and coworkers[5] reported beneficial results from administration of liberal quantities of B vitamins in pigs showing symptoms of a chronic enteritis.

Observations reported in this paper are concerned primarily with the therapeutic value of nitrofurazone† N.N.R. (5-nitro-2-furaldehyde semicarbazone), which is an entirely different type of chemical compound from the sulfonamide group of drugs. Work was conducted under controlled conditions, using a single virulent strain of *Sal. choleraesuis* obtained from the Pathological Division of the Bureau of Animal Industry. The virulence of this culture was maintained by lyophilization. Briefly, the process of lyophilization was accomplished by rapidly freezing the culture at a very low temperature and drying under a high vacuum while in the frozen state.

Materials and Methods

Pigs were raised on the experimental farm or purchased from farmers in the vicinity. With the exception of experiment 2, all of the animals in any one experiment were obtained from the same source. In the case of the one

*Dr. Hess and Clark, Ashland.

†Manufactured by Eaton Laboratories, Norwich, N. Y.

exception, pigs were purchased from neighboring farmers who had used the same herd sire on sows which were closely related.

Pigs were confined in small pens and after a few days to accustom them to the change in quarters, they were weighed individually and divided into groups of approximately the same average weight. Temperature of each animal was recorded daily for approximately a week prior to infection and for at least two weeks thereafter. All received a 20% protein ration containing a feed supplement fortified with vitamin B_{12} and antibiotics, at a level which investigators in the field of nutrition have recommended. Each animal was weighed on the day of infection and at weekly intervals for three to four weeks thereafter. At this time all groups were confined together. Each pig was again weighed ten to 12 weeks after infection, at which time the experiment was terminated.

All subjects were infected with a 24-hour nutrient broth culture of *Sal. choleraesuis* freshly reconstituted from the lyophilized state for each experiment. Each animal in the first four experiments was fed 25 cc. of the culture in a small quantity of milk on two successive days. In experiment 5, the quantity of culture was reduced to 0.3 cc. for each pig for two days.

Drugs were suspended in a hydrolyzed cornstarch solution, or a solution of carboxy methyl cellulose, and administered into the esophagus by means of a short stiff rubber tube connected to the end of the long nozzle of an ordinary dose syringe. For administration in the feed, the drug was first premixed with a small quantity of feed and then incorporated into a larger quantity and mixed for several minutes in a horizontal type mixer.

DATA

Experiment 1

Fifteen pigs were divided into three lots of five pigs each. Each was fed 25 cc. of a 24-hour broth culture of *Sal. choleraesuis* in a small quantity of milk for two consecutive days. Data on these animals are presented in table 1.

TABLE 1. Effectiveness of Nitrofurazone and Bacitracin for Controlling Sal. choleraesuis Enteritis in Young Pigs (Note: Each pig was fed 25 cc. of a 24-hour broth culture of a virulent strain of Sal. choleraesuis on 5/29/50 and 5/30/50)

Lot No.	Pig No.	Temperature Above 104 F.		Treatment	Weight (Pounds)						Mean Daily Gain (lb.)	Date of Death 1950
		No. of Days	Max.		at infection	1 Wk.	2 Wk.	3 Wk.	4 Wk.	11 Wk.		
	6	6	107.0		35.5	27.0						6/7
	7	5	106.6	None	33.5	27.5	26.0	30.0	34.0	106.5	0.94	
1	8	7	107.4		42.5	33.0						6/8
	9	4	106.6		31.5	24.0	20.0	23.0	25.0	76.5	0.58	
	18	8	107.6		26.0	23.0						6/8
	11	4	106.4	30,000 units BTN* b.i.d.	32.0	34.0	34.0	32.0	23.0			6/30
	16	3	106.8	to each 5/31 through 6/1	42.0	43.0	50.0	56.0	65.5	147.0	1.36	
2	17	6	108.5	0.3 gm. NFZ** b.i.d. to	33.0	33.5	39.5	47.5	59.0	151.5	1.53	
	19	4	107.6	each 6/2 through 6/3	27.0	26.0	32.5	38.0	43.5	102.0	0.97	
	20	5	107.6	0.05% in feed 6/8 through 6/11	37.5	34.0	42.0	47.0	52.0	109.5	0.93	
	1	1	107.4	0.3 gm. NFZ b.i.d. to	36.0	38.0	42.0	47.0	52.0	126.0	1.16	
	2	2	106.6	each 5/31 through 6/1	42.0	46.5	49.0	55.5	61.5	128.5	1.12	
4	3	5	108.2	0.15 gm. to each 6/2	43.0	44.5	49.0	60.0	68.0	166.5	1.6	
	4	2	107.2	0.02% in feed 6/6 to 6/11	28.0	26.5	31.5	39.5	46.0	106.5	1.01	
	5	1	107.6		29.0	32.5	38.0	44.0	48.0	104.5	0.98	

*Bacitracin; **Nitrofurazone

Thirty-six to 48 hours after the first infective material was fed, all pigs were visibly sick. They lay in the bed most of the time and it was difficult to get them to move about. They exhibited little or no interest in feed but drank considerable water. A few animals vomited when they were forced to move about. By the third day most were scouring profusely and none of them was eating.

Three of the five untreated controls in lot 1 died. One died on the eighth day and the other two on the ninth day following infection. At necropsy, the wall of the cecum and colon was somewhat thickened and edematous, and the small lymph follicles on the peritoneal surface were enlarged. In one animal there was a deposition of fibrin over the peritoneal surface of the cecum. Mucosa of both the

cecum and colon was almost completely covered with a thick diphtheritic membrane, or as it has been so aptly described, with a "cork lining". There was considerable congestion of the fundus and pyloric region of the stomach. All three animals were very thin and badly dehydrated at the time of death. Four weeks after infection the two survivors were still in poor condition. At eleven weeks after infection both animals were still living, but one of them had gained poorly.

Each animal in lot 2 was given 30,000 units of bacitracin†† twice daily for two days, beginning on the first postinfection day. This treatment was ineffective, and the condition of the animals rapidly deteriorated. On the third day after infection, each pig was dosed individually with 0.3 gm. of nitrofurazone twice daily for two days. After the first day of treatment, the body temperature was decreased and the pigs began to eat sparingly. The character of the feces improved and by the third day after treatment they were nearly normal. On the fifth day after treatment was discontinued, two of the animals showed a slight rise in temperature and the feces became partially fluid in character. As the appetite was good, they were put on a medicated feed containing 0.055% nitrofurazone for four days. During this time they consumed 12.6 gm. of nitrofurazone, or approximately 0.6 gm. of the drug per pig daily. The character of the feces improved after this treatment. Four of the pigs completely recovered and made a normal growth during the 11-week observation period. One animal (11) relapsed after the third week and eventually died. At necropsy, lesions were similar, but not as extensive, as those seen in the untreated controls.

Each pig in lot 3 was dosed with 0.3 gm. of nitrofurazone twice daily for two days following infection. Treatment was initiated on the first postinfection day. On the third day of treatment each animal received only one 0.15 gm. dose. At this time, the temperature of all pigs had returned to the normal range, and all were eating well by the fifth day after infection. On the sixth day, two pigs evinced a slight rise in temperature and at this time feed containing 0.02% of the drug was given. The medicated feed was continued for six days, during which time each pig consumed approximately 0.25 gm. of nitrofurazone daily. All five animals recovered and made normal gains during the feeding-out period.

TABLE 2. Effect of Nitrofurazone on Sal. choleraesuis Enteritis in Young Pigs
(Note: Each pig was fed 25 cc. of a 24-hour broth culture of a virulent strain of Sal. choleraesuis on 7/17/50 and 7/18/50)

Lot No.	Pig No.	Temperature Above 104 F.		Treatment	Weight (Pounds)						Mean Daily Gain (lb.)	Date of Death 1950
		No. of Days	Max.		at infection	1 Wk.	2 Wk.	3 Wk.	4 Wk.	11 Wk.		
	1	8	108.2		39.0	29.5	24.0					8/6
	7	7	107.8	None	31.5	24.5	20.0	16.5				8/11
1	11	3	106.4		39.5	33.5	38.0	47.5	56.5	114.0	1.06	
	16	4	108.2		36.0	32.5	36.5	40.5	46.0	94.0	0.82	
	5	3	107.0	0.3 gm. b.i.d. to each	37.5	38.5	46.0	52.5	62.5	121.5	1.2	
	18	2	105.2	7/20 through 7/22	32.5	31.5	36.5	43.5	49.5	91.0	0.83	
2	19	4	106.4	0.033% in feed 7/27	28.5	24.0	29.0	36.5	45.5	92.0	0.9	
	20	3	106.0	through 7/29	32.0	32.0	39.5	45.0	53.5	113.0	1.15	

Experiment 2

In this test, there were two lots of four pigs, each of which was fed 25 cc. of the infective culture for two consecutive days. Results of this test are summarized in table 2.

The four animals in the first lot received no treatment after infection. Two died, and at necropsy, lesions were present similar to those described above for the untreated controls in experiment 1. Remaining animals were off feed and had a severe diarrhea for four days, after which time they recovered rather promptly. Both animals made a fairly good gain during the feeding-out period.

Each pig in lot 2 was given 0.3 gm. of nitrofurazone twice daily for three days, beginning on the second day after infection. Response to treatment was good in that they returned to full feed and the character of the feces improved from day to day. Due to a misunderstanding on the part of the caretaker this lot was given 0.033% of the drug in the feed for three days, beginning on the ninth day after infection. All pigs survived and made reasonably good gains for 11 weeks following infection. There was no recurrence of the disease noted during this time.

††Supplied by Commercial Solvents Corporation, Terre Haute, Ind.

Experiment 3

In this experiment, there were two lots of five pigs. Each pig was fed 25 cc. of the infective culture for two consecutive days. Data for this experiment appear in table 3.

All of the untreated control animals in lot 1 had died by the fourth week after infection. There was inappetence and severe scouring up to the time of death. Necropsy findings were similar to those already described for the untreated controls in previous experiments.

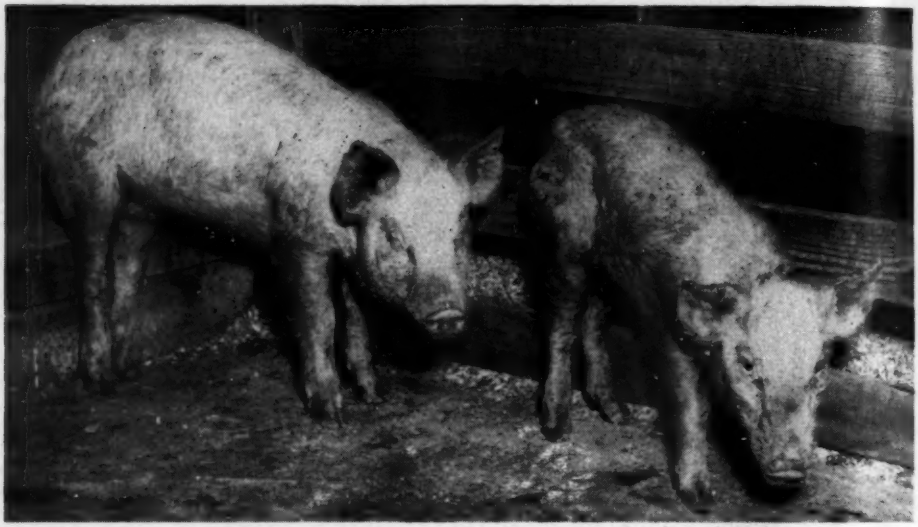

Fig. 1. Appearance of two untreated pigs four weeks after infection with 0.3 cc. of a 24-hour nutrient broth culture of a virulent strain of *Sal. choleraesuis* preserved by lyophilization. At this time, the pig on the left weighed 11 lb. less than than it did at the time of infection. The animal on the right lost 16 lb. during the same period.

Each animal in lot 2 received a different dosage of nitrofurazone, beginning on the first day following infection. This was done in an attempt to ascertain the minimum curative dosage of the drug.

Pig no. 1 received 0.1 gm. of the drug twice daily for three days, followed by 0.1 gm. once daily for seven days. For the first few days after treatment was initiated, this pig appeared to be responding to treatment, but a few days later it was observed to be off feed. The animal gradually lost condition and died 22 days after infection. At necropsy, the mucosa of the large bowel appeared normal. There was a slight congestion of the fundus area of the stomach. The only significant lesion encountered was a necrosis of the tissues in the region of the pharynx. Evidently faulty technic in the numerous administrations of the drug resulted in injury to the pharyngeal region and infection with saprophytic organisms which terminated fatally.

Pig no. 5 received 0.15 gm. of the drug twice daily for three days, followed by the same quantity once daily for three days. This pig was very small at the time of infection, weighing only 15.5 lb., and was considered to be a poor subject. The response to treatment was satisfactory in that the appetite returned to normal and scouring had subsided by the fifth day of treatment. This animal gained slowly, but in view of the fact that it was retarded in its development prior to infection, it appears that the drug effectively checked the course of the disease.

Pig no. 29 was given 0.2 gm. twice daily for three days. The same dosage was continued once daily for three more days. The appetite remained poor and scouring continued intermittently up to the time of death, three weeks after infection. At necropsy the mucosa of the cecum and colon was almost entirely covered by a diphtheritic membrane.

Pig no. 3 received 0.25 gm. twice daily for three days, followed by one 0.25 gm. dose for three days. This animal responded well to this dosage and completely recovered, as indicated by the mean daily gain over a ten-week period after infection.

Pig no. 7 was given 0.3 gm. of the drug twice daily for three days followed by one 0.3 gm. dose on each of the next three days. Symptoms subsided promptly but a moderate degree of scouring recurred during the second week after infection. The diarrhea disappeared after a few days, but the animal gained weight slowly, which suggests that a subclinical infection persisted, or that it suffered irreparable damage from the infection.

TABLE 3. Effect of Nitrofurazone on Sal. choleraesuis Enteritis in Young Pigs

(Note: Each pig was fed 25 cc. of a 24-hour broth culture of a virulent strain of Sal. choleraesuis on 4/9/51 and 4/10/51)

Lot No.	Pig No.	Temperature Above 104 F.		Treatment	Weight (Pounds)					Mean Daily Gain (lb.)	Date of Death 1951
		No. of Days	Max.		at infection	1 Wk.	2 Wk.	3 Wk.	10 Wk.		
	4	4	108.4		44.5	31.5	24.5				4/26
	13	8	107.2		42.0	38.5	31.5	30.0			5/23
1	16	4	107.8	None	26.0						4/14
	22	5	105.7		34.0	27.0	21.0	17.0			5/1
	26	4	107.4		33.0	23.5					4/17
	1	1	107.2	0.1 gm. b.i.d. 4/11 through 4/13 0.1 gm. 4/14 through 4/21	29.0	25.5	21.0	17.0			5/3
	5	1	107.0	0.15 gm. b.i.d. 4/11 through 4/13 0.15 gm. 4/14; 4/19 through 4/21	15.5	17.0	20.5	25.5	67.0	0.73	
2	29	2	106.4	0.2 gm. b.i.d. 4/11 through 4/13 0.2 gm. 4/14; 4/19 through 4/21	31.0	29.0	29.5	26.0			6/1
	3	2	108.0	0.25 gm. b.i.d. 4/11 through 4/13 0.25 gm. 4/14; 4/19 through 4/21	39.0	41.5	48.0	57.5	134.5	1.36	
	7	2	107.2	0.3 gm. b.i.d. 4/11 through 4/13 0.3 gm. 4/14; 4/19 through 4/21	44.5	47.5	50.0	50.0	94.0	0.7	

Experiment 4

In this test, 15 pigs were divided into five lots of three pigs each. Each animal was fed 25 cc. of the infective culture on two consecutive days. The comparative effectiveness of nitrofurazone, sulfathalidine and sulfaguanidine for controlling the infection in this group appears in table 4.

None of the untreated pigs in lot 1 survived. At necropsy, lesions were encountered similar to those already described for the infected untreated animals in the previous experiments.

In lot 2, each pig was given 0.25 gm. of nitrafurazone twice daily for four days. Treatment was initiated on the second day after infection. By the second day of treatment the appetite improved and the character of the feces returned to normal on the fourth day. No further evidence of the disease appeared after treatment was discontinued, and all animals gained well during the ten-week observation period.

In lot 3, each animal was given 0.3 gm. of nitrafurazone twice daily for four days, beginning on the second day after infection. On the second day of treatment there was improvement in the appetite and by the third day the feces were nearly normal in appearance. All three of these animals survived and gained well during the ten-week period following infection.

The pigs in lot 3 were each given 1.5 gm. of sulfathalidine twice daily for four days. After this course of treatment all three animals were gaunt, scouring and the appetite subnormal. During the next three days there was no improvement in their condition, and the same treatment was repeated for two additional days. One pig (no. 19) showed little

response to treatment. Diarrhea continued for several weeks and the appetite was poor. Ten weeks after infection this animal had gained only a few pounds. The other two animals continued to scour for several days after treatment was discontinued, but by the third week after infection they appeared to have recovered. Both of the latter animals gained well during the 12-week period after infection.

reduced in an effort to produce a less severe type of disease similar to that encountered in the field. Data on these two lots are contained in table 5.

Four of the five untreated controls in lot 1 were living ten weeks after infection. One animal (no. 5) died during the sixth week. At necropsy there were a few patches of diphtheritic inflammation scattered over the

TABLE 4. Comparative Effect of Nitrofurazone, Sulfathalidine and Sulfaguanidine on Sal. choleraesuis Enteritis in Young Pigs

(Note: Each pig was fed 25 cc. of a 24-hour broth culture of a virulent strain of Sal. choleraesuis on 6/27/51 and 6/28/51)

Lot No.	Pig No.	Temperature Above 104 F.		Treatment	Weight (Pounds)						Mean Daily Gain (lb.)	Date of Death 1951
		No. of Days	Max.		at infection	1 Wk.	2 Wk.	3 Wk.	4 Wk.	12 Wk.		
	9	5	107.6		23.0	17.0						7/7
1	16	8	107.6	None	36.5	31.5	26.5	21.5				7/23
	22	6	107.0		45.5	37.0						7/13
	33	3	107.4	0.25 gm. NFZ* b.i.d. to	24.0	29.0	37.0	41.5	48.0	134.5	1.31	
2	11	4	108.0	each 6/30 through 7/3	32.5	37.5	45.0	53.5	60.5	161.5	1.53	
	27	4	108.2		45.0	51.0	60.5	65.5	72.0	170.5	1.49	
	29	4	107.8	0.3 gm. NFZ b.i.d. to	35.5	41.5	50.0	59.0	65.0	158.5	1.46	
3	3	4	107.6	each 6/30 through 7/3	40.4	45.0	57.0	66.0	75.0	180.0	1.66	
	24	4	108.4		42.5	48.0	54.5	62.0	71.5	155.5	1.34	
	19	4	107.4	1.5 gm. STD** to each	28.5	24.0	23.5	21.0	22.5	55.0	0.31	
4	6	2	106.8	6/30 through 7/3; 7/7	31.0	31.5	37.0	42.0	48.0	145.0	1.35	
	12	6	108.2	through 7/8	41.5	38.5	39.5	46.5	54.0	152.0	1.31	
	20	5	107.4	1.5 gm. SGN*** b.i.d. 6/30 through 7/3; 7/7 through 7/8	36.5	37.5	44.0	50.0	56.0	149.5	1.34	
5	34	2	106.8	2.0 gm. SGN b.i.d. 6/30 through 7/3; 7/7 through 7/8	38.5	39.0	45.0	50.5	56.5	155.5	1.39	
	28	4	107.0	1.5 gm. SGN b.i.d. 6/30 through 7/3; 7/7 through 7/8	36.0	33.0	42.0	44.5	43.0	120.5	1.0	

*Nitrofurazone; **Sulfathalidine; ***Sulfaguanidine

Lot 5 was treated with sulfaguanidine. Two of the pigs (no. 20 and 28) were each given 1.5 gm. of the drug twice daily and the other (no. 34) 2 gm. twice daily, for four days. The appetite improved, but scouring continued and the general condition of all animals deteriorated during the next three days. Treatment was again instituted and continued at the same dosage for two days. One pig (no. 28) scoured periodically for four weeks after medication was discontinued, and up to the tenth week appeared less thrifty than the other two.

Experiment 5

Ten pigs were confined in two lots of five each. Each animal was fed only 0.3 cc. of a 24-hour culture of *Sal. choleraesuis* on two consecutive days. The amount of culture was

mucosa of the cecum and colon. One of the survivors (no. 1) gained only 15.5 lb. over the ten-week period after infection. Two of the remaining three animals did not gain at a normal rate, although they appeared thrifty.

Each pig in lot 2 was given 1 gm. nitrofurazone on the second day and 0.5 gm. on the third day after infection. The drug was given in a single dose on each day. All animals were living ten weeks after infection and four had gained weight normally during this period. Pig no. 19 failed to gain well which indicates that it probably should have received further treatment.

Toxicity Studies

The effect of large doses of nitrofurazone has been observed in six normal pigs. The drug was administered in the feed or given

orally suspended in a hydrolyzed cornstarch solution.

A 30-lb. pig was given 2.05 gm. (150 mg. per kg. of body weight) suspended in a hydrolyzed cornstarch solution. No evidence of intoxication was shown by this animal.

A 34-lb. animal was given 3.15 gm. (200 mg. per kg. of body weight) and likewise showed no toxic symptoms.

A single dose of 8.8 gm. was administered by stomach tube to a 64-lb. pig (300 mg. per kg. of body weight). There were inappetence and lethargy for two days and on the third day there were incoordination and weakness of the legs. By the following day the animal was unable to get to its feet. Some water was consumed but inappetence persisted. From the fifth to the eighth day the animal was cyanotic but after this time the condition improved and small quantities of feed were consumed. There was an almost complete motor paralysis of the legs until the 14th day, at which time the animal was able to stand unassisted for short periods. Incoordination and weakness of the legs persisted until death occurred 25 days after treatment. At necropsy, there was an intense congestion of the meninges. Microscopic examination of tissues from this animal revealed degeneration of the cortex and cord neurons. There were several areas of congestion in the kidney with degeneration of the cells of the convoluted tubules. Diffuse cloudy swelling was present in the pancreas.

A single dose of 9.3 gm. was given to a 52.5-lb. pig (400 mg. per kg. of body weight). The following day this animal was active and the appetite was about normal, but there was a slight stiffness in the movement of the pelvic limbs. On the second day there was a partial paralysis of the hind legs. Death occurred on the third day. At necropsy the subcutaneous blood vessels were congested. There were numerous petechial hemorrhages over the surface of the thymus and the meninges were intensely congested. The liver had a mottled appearance and the kidneys were congested.

A 30-lb. pig was given 2.76 gm. of the drug in the feed during one day. On each of the next two days 1.38 gm. was consumed in the feed. The feces became quite firm and the urine was dark brown in color. Fifteen days later the same animal was given 5.9 gm. of the drug daily for two days in the feed. The appetite was depressed temporarily. Four days later 5.9 gm. was again given in the feed. There were inappetence and incoordination in the movement of the pelvic limbs over a period of four days, after which time the animal returned to normal.

A 60-lb. pig received 5.4 gm. of the drug in the feed for one day. On the next day 3.7 gm. were consumed in the feed. For the next two days the appetite was impaired and the animal was lethargic. On the fourth day after the initial dose, 5.4 gm. of the drug were consumed in the feed. The following day the animal was completely off feed. Two days later 5 gm. were administered by stomach tube and the same dosage repeated on the following day. For the next 48 hours there were inappetence and marked incoordination of the pelvic limb movements. Two days later the animal was eating and manifested no further evidence of intoxication.

TABLE 5. Effect of Nitrofurazone on Sal. choleraesuis Enteritis in Young Pigs
(Note: Each pig was fed 0.3 cc. of a 24-hour broth culture of a virulent strain of Sal. choleraesuis on 10/10/51 and 10/11/51)

Lot No.	Pig No.	Temperature Above 104 F.		Treatment	Weight (Pounds)						Mean Daily Gain (lb.)	Date of Death 1951
		No. of Days	Max.		at infection	1 Wk.	2 Wk.	3 Wk.	4 Wk.	10 Wk.		
	1	2	106.0		34.5	32.5	31.5	33.5	36.5	50.0	0.22	
	5	3	107.6	None	28.0	28.0	31.0	35.5	37.5			11/23
1	8	3	106.8		27.5	28.0	32.0	36.0	41.0	87.5	0.85	
	13	3	106.6		33.5	31.0	28.5	31.5	36.0	87.0	0.76	
	21	5	107.0		44.5	43.0	41.0	48.5	53.0	115.5	1.01	
	3	1	106.8		45.5	44.5	51.0	55.5	62.0	118.5	1.04	
	12	2	106.2	1.0 gm. to each on 10/13	27.0	25.5	34.5	40.5	45.5	101.0	1.05	
2	15	2	107.0		36.0	35.0	44.5	52.0	58.5	119.5	1.19	
	19	6	106.2	0.5 gm. to each on 10/14	28.0	28.5	33.0	36.0	38.0	65.0	0.52	
	23	1	106.0		32.5	34.0	42.5	50.5	58.5	118.5	1.22	

Discussion and Summary

The studies herein reported corroborate the findings of Schoening, *et al.*, that when pigs are fed a virulent culture of *Sal. choleraesuis* preserved by lyophilization, there is produced

a disease with symptoms and lesions indistinguishable from the enteric condition commonly diagnosed as necrotic enteritis in the field. In general, the severity of the disease follows the quantity of culture fed. It is, therefore, possible to conduct a critical examination of the therapeutic value of drugs for treating this important disease under controlled conditions.

Five controlled experiments involving 58 susceptible pigs have been conducted. In the first four tests, the pigs were each infected with 25 cc. of a 24-hour nutrient broth culture for two consecutive days. Such a dosage produced a severe type of disease as 13 out of 17 untreated animals died. Nineteen pigs were treated twice daily with 0.25 to 0.3 gm. of nitrofurazone for two to four days. Only one of these 19 animals died after treatment.

Six pigs were treated with the commonly recommended, poorly absorbed sulfonamides, three with sulfaguanidine and three with sulfathalidine. These recovered but they required a longer period of treatment than did those receiving nitrofurazone. However, the number of animals involved was too few to permit adequate comparison.

In an attempt to determine the minimum dosage of nitrofurazone necessary for control of the severe type of the disease, six pigs were treated twice daily with quantities varying from 0.1 to 0.3 gm. of the drug. The results indicate that 0.25 to 0.3 gm. given morning and evening, or a total of 0.5 to 0.6 gm. daily is the optimum dosage.

When the quantity of culture was reduced to 0.3 cc. for two days the disease was more nearly comparable to that seen in the field. One out of five untreated animals died and another pig was permanently stunted from the effects of the disease. In a herd which has recovered from the disease, as it occurs in the field, there are usually several animals which fail to recover completely and are never profitable from the standpoint of conversion of feed into meat.

A single dose of 1 gm. of nitrofurazone followed by 0.5 gm. on the next day effectively controlled the disease. Some animals may require additional treatment as one out of five pigs receiving the drug at this dosage showed a poor rate of gain. Possibly treatment should be continued by administering low levels of the drug in the feed.

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Vesicular Exanthema

Considerable space recently has been given in the daily press in reference to a vesicular disease of swine clinically indistinguishable from foot-and-mouth disease. Vesicular exanthema has been identified in midwestern and California droves.

This is an acute febrile condition of swine in which confluent vesicles appear on the soles, interdigital tissue and the coronary region of the claws. Severe lameness may result from necrosis of the affected parts. Vesicles often form on the snout, nose, lips, mucous membranes of the mouth, and on the udder of nursing sows. Since lesions of foot-and-mouth disease, vesicular stomatitis, and vesicular exanthema are identical, animal inoculation tests are necessary for diagnosis. Characteristics of the causative virus which identify the condition follow a charted course in various test animals. These were described by Dr. J. Traum in 1934. From this work, the disease was demonstrated to be a specific entity.

Death occurs in suckling pigs. Mortality in older animals is low. However, in severe outbreaks morbidity rate may be extreme. Losses result from condemnations, loss of weight, and abortions.

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Good professional service is the best weapon against illegal veterinary practice.

Anthrax In Livestock During 1951 And Comparative Data On The Disease From 1945 Through 1951

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IN previous reports, statistical data were presented on the national incidence of anthrax in livestock from 1945 to 1950 inclusive[1,2,3,4]. During this six-year period, 658 anthrax outbreaks were reported from 32 states with estimated losses of 8,504 head of livestock. Occurrences in new areas were reported from 51 counties in 16 states.

The data presented in this report, as in previous ones, are based principally on information furnished by Bureau veterinarians in charge of field work in various states after consultation with livestock sanitary officials. Because of the difficulty in obtaining precise and complete information, as in all national surveys of this nature, the data presented cannot be considered 100% accurate. However, facts and figures are believed to represent a comprehensive picture of the national incidence of the disease for the period covered.

Table 1 shows the incidence of anthrax and loss of livestock (cattle, horses, mules, sheep, and swine) in each state reporting outbreaks during 1951. Tables 3 and 4 give comparable data for the seven-year period from 1945 to 1951 inclusive. To present a general picture of the geographic distribution of the disease, a map of the United States showing the number of outbreaks reported from 1945-1951 inclusive and the location of the counties in which the outbreaks occurred is included.

1951 Survey

Data compiled from the 1951 survey indicate that during the year 483 anthrax outbreaks were reported from 25 states involving 113 counties with a total loss of 2,753 head. It is apparent from table 4 that a considerably greater number of these occurred in 1951, involving more counties than in any single year from 1945 through 1950. However, the actual losses in animals was not as high as in 1946, when there occurred heavy losses during severe epizootics of the disease in Louisiana. No major widespread outbreaks were reported last year.

The greatest losses were reported from infected areas in Missouri where there was a total of 694 deaths, of which 440 were in horses and mules. Fairly large losses were reported by California, Florida, Illinois, Iowa, Kentucky, Nevada, Tennessee, and Texas.

California was the only state which reported anthrax in sheep. In an outbreak involving approximately 2,000 head of sheep, 30 were reported to have died.

Anthrax Outbreaks in Swine

An unusual feature of the disease as it occurred during 1951, was the great number of widely scattered outbreaks in swine. Data covering losses for 1951 show that the total number of reported deaths was 1,088 as compared with a total of 1,001 deaths in cattle. The greatest losses in this species were reported by Illinois (279), Iowa (281), Kentucky (131), and Missouri (147). Coincident with the increased number of cases of anthrax in swine is the condemnation of 232 infected carcasses by establishments operating under federal meat inspection.

Although swine are considered to be far more resistant to anthrax than other species of livestock, the disease is by no means uncommon in areas in the United States where the infection exists enzootically. Although in non-infected areas sporadic outbreaks may occur in swine as a primary infection from consumption of contaminated feed without involvement of other livestock, it usually follows the appearance of the disease in other animals on the farm. Thus it most commonly occurs in swine from feeding on the carcass of an animal dead of anthrax, from following infected cattle during an outbreak, or by deep rooting on infected pastures during the spring season. While the disease may occur in swine in an acute or chronic form with characteristic clinical symptoms and be recognized easily, many cases where it occurs in the less conspicious mild, chronic or latent forms probably are not detected.

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Of special significance and interest were the primary outbreaks that occurred principally in Illinois, Iowa, Indiana and Ohio on premises hitherto considered free from infection and far removed from recognized anthrax areas.

Since the epizootiologic backgrounds of occurrence in swine in these four states appeared to be somewhat similar, a special effort was made by the BAI to determine the source of infection. To this end the Bureau's field veterinarians in charge, in cooperation with the state livestock sanitary officials, were directed to obtain specific information on the history and circumstances connected with each outbreak.

However, even though all possibilities were explored these investigations failed to establish a definite source of infection. The history connected with some suggested that contaminated mixed feed may have been the source of infection. Examination of suspected samples of such feed failed to reveal contamination with *Bacillus anthracis*.

Resume of Outbreaks Reported in Swine for 1951

In Illinois, from August 16 to December 26, 1951, 12 outbreaks in swine occurred in ten different counties located in the southern and central parts of the state. In most instances, these occurred in widely separated areas heretofore regarded as noninfected territory and under varying herd management and sanitary conditions. The disease was also peculiar in its behavior in that swine in most instances were the only animals affected. Most of the cases were in sows and pigs and, in general, losses were small. In a number of instances, cattle and other livestock on the same farm were not affected. The possibility that contaminated feed supplements may have been the source of the infection was investigated with negative results. Although spread over a large territory, no evidence was found to indicate that infection spread from farm to farm. The disease occurred in swine on timber pastures, on sandy soil, on rolling land, on flat black land, and on river bottom pastures, and in some instances, on low land subject to flooding.

In Iowa during 1951, 14 sporadic outbreaks in swine were reported from 12 different counties heretofore regarded as noninfected territory. In most cases, losses were small and in no instance did the disease spread from infected to noninfected herds. The source of the infection in these outbreaks was not determined. It was reported that some of the pastures used had considerable standing water on them resulting from an unusual rainy season.

In Ohio during September 1951, a sharp sporadic outbreak occurred in Preble County

TABLE 1. States Reporting Anthrax Outbreaks in Livestock in 1951 and Data on Incidence of the Disease

State	No. of counties	No. of outbreaks	Losses from Anthrax				Total livestock losses
			Cattle	Swine	Horses & Mules	Misc.	
Alabama	1	1	2				2
Arkansas	4	4	6		2		8
California	10	18	168	3		30 sheep	201
Colorado	1	1	5				5
Florida	3	21	206		3	13 deer	209
Illinois	11	14	8	279			287
Indiana	1	1		4			4
Iowa	14	16	8	281			289
Kentucky	2	12	18	131	44		193
Louisiana	14	15	82				82
Minnesota	3	3	4	13			17
Mississippi	2	2	3				3
Missouri	7	150	107	147	440		694
Nebraska	3	3	3	4			7
Nevada	2	30	115	40			155
New Jersey	1	4	15				15
New York	1	1	1				1
North Dakota	1	1	3	2			5
Ohio	1	1		50			50
Oklahoma	2	5	39				39
Oregon	2	5	14				14
South Dakota	6	7	22	9			31
Tennessee	3	140	75	45	145		265
Texas	15	25	92	80			172
Wisconsin	3	3	5			102 mink	5
Total	113	483	1001	1088	634	30 (sheep)	2753*

*Mink and deer not included in total livestock losses.

in a herd of approximately 200 animals. Fifty pigs about four weeks of age succumbed to the disease. This was the first reported outbreak in Ohio in more than 30 years, and caused a great deal of apprehension. The source of infection was not determined. Laboratory examination for the presence of *Bacillus anthracis* in prepared feed supplements fed to the herd was carried out by the Ohio State University with negative results.

In Indiana during August 1951, a small sporadic outbreak was reported from Vermillion county. Although contaminated feed was suspected, the origin of infection was not definitely determined. Indiana, like Ohio, is one of the states that in the past has reported few cases of the disease.

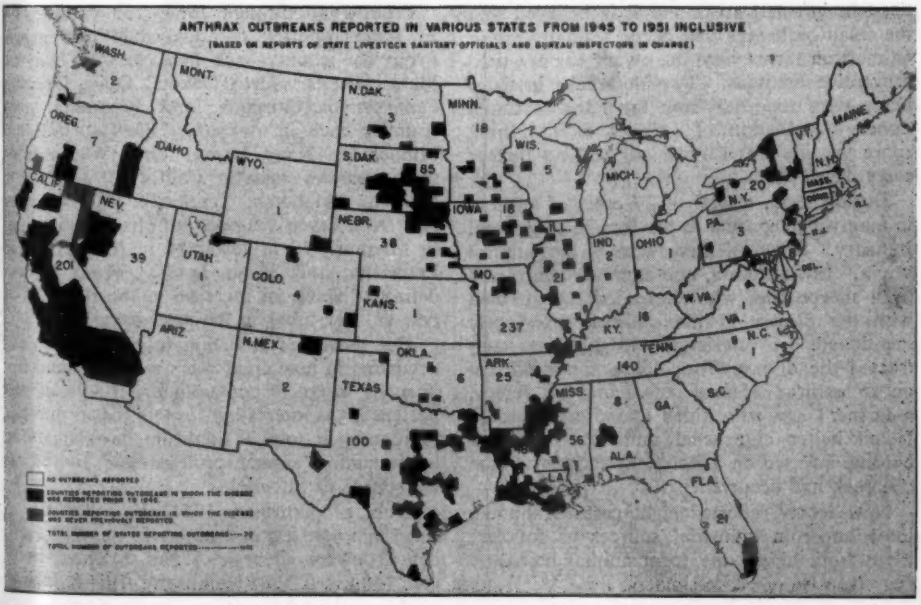
In Nevada, 11 outbreaks in swine with a loss of 40 head were reported in 1951. Five of the 11 occurred on ranches that had no cattle. In some of the affected herds, losses were small and it was the opinion of investigators that at least some of these resulted from fly infestation.

A number of additional outbreaks in swine were reported from other states but the majority of these occurred on infected premises and in connection with diseased cattle, horses or mules.

In 1951, outbreaks in 51 counties which had no recorded previous history of the disease were reported as follows: From one county each in Indiana, Kentucky, Mississippi, New Jersey, and Ohio; two counties each in Florida, Minnesota, and Oklahoma; three counties each in Louisiana, Tennessee, and Wisconsin; four in Missouri; six in Texas; ten in Illinois; and 11 in Iowa. Outbreaks were reported from Florida, Ohio and Tennessee which had not reported cases since prior to 1944. Several states showed a noticeably greater number of outbreaks and larger losses in the one year of 1951 than the total numbers for the preceding six-year period. (See table 2.)

Incidence of infection in Florida in 1951, was the first that has been reported from that state in more than 20 years. The disease involved both beef and dairy cattle and the source of the infection was not definitely determined although in one instance, bone meal was suspected. Buzzards were believed to be one of the factors contributing to the spread, so a trapping and elimination program was put into effect in addition to strict quarantine of infected premises and vaccination of all cattle within and surrounding the quarantined areas.

A number of the anthrax outbreaks in Tennessee occurred in the northern part of Lake county, an area subject to flooding every few years. In the extreme southeastern part of Missouri, on the west bank of the Mississippi River, there is an area of recognized anthrax infection. On the east bank of the Mississippi, in this same area, outbreaks occurred in recent years in Kentucky and Tennessee.



Of special interest is the history of infection in mink and deer. In Wisconsin, 102 mink died. The source of the infection was suspected to be infected animal food. Thirteen deer in Florida were reported to have died on premises where outbreaks in cattle had occurred. State officials reported that anthrax bacilli were isolated from the bones of three of these deer.

Incidence of Anthrax in Territories and Possessions

Alaska reports that there were no cases of anthrax during 1951. In Hawaii no cases of anthrax are known to have occurred since 1937. One premises on the Island of Oahu and one on the Island of Kaui are considered possible infected areas. Cattle and horses on these premises are vaccinated annually with anthrax spore vaccine. Six outbreaks were reported from Puerto Rico with an estimated loss of 18 cattle. Yearly vaccination is practiced in infected areas and it is believed that the small outbreaks that occur annually are in animals on farms were the owner has not used preventive methods. Two outbreaks in dairy herds were reported from the Panama Canal Zone. Vaccination of animals with anthrax spore vaccine was carried out and no further cases developed.

TABLE 2. Outbreaks and Losses. Six-Year Period 1945-50 Compared with Those in 1951

State	Period 1945-1950 inclusive		Year 1951	
	Outbreaks	Losses	Outbreaks	Losses
Florida	0	0	21	209
Illinois	7	23	14	287
Iowa	2	5	16	289
Kentucky	4	35	12	193
Missouri	87	404	150	694
Nevada	9	63	30	155
Ohio	0	0	1	50
Tennessee	0	0	140	265

During 1951, a total of 17 cases of anthrax in man were reported to the Bureau of Animal Industry. Seven of these were of agricultural origin. In California, one case was contracted by a sheepherder who sheared sheep affected with the disease in the outbreak previously mentioned. A veterinarian in this state contracted the disease from doing a postmortem on an anthrax-infected cow. In New Jersey, a farmer's son who killed and dressed an infected heifer contracted anthrax. A tenant became infected on a Kentucky farm where an outbreak had occurred.

New Jersey reported eight cases of human infection from industrial sources as follows: three from hides, one from animal hair, and four from wool processing.

During the outbreak in the southern part of Florida in 1951, the disease was acquired by five persons: a cowboy who skinned a cow that had died of anthrax, two veterinarians who vaccinated exposed cattle in the area, a child of the nurse who attended these veterinarians, and a laboratory technician who handled a suspected specimen.

Records of the U. S. Public Health Service for 1951 give a preliminary total of 60 cases of anthrax in man[5].

Anthrax Outbreaks from 1945 Through 1951

Table 3 indicates that during this seven-year period, one or more outbreaks were reported from each of 35 states. During this period, there were 1,141 outbreaks and a total loss of 11,257 head of livestock.

Livestock losses were heaviest in California, Louisiana, Missouri and Texas. Most of the outbreaks were sporadic and resulted in minor losses. Louisiana was the only state that reported widespread and severe infection. Epizootics of this nature occurred there in 1946 and 1948.

Missouri, which prior to 1945 had reported only isolated occurrences, showed a marked increase in the prevalence of the disease. During the seven-year period 237 outbreaks with a livestock loss of 1,098 were reported, of which 150 outbreaks with 694 deaths occurred in 1951.

From 1945 through 1951, 133 counties in 27 states reported the disease in new areas. From one county each in Colorado, Kansas, Massachusetts, North Dakota, Ohio, Oregon, Pennsylvania, Virginia, and Wyoming; two counties each in Arkansas, Florida, Indiana, Minnesota, New Jersey, and Washington; three counties each in California, Kentucky, Nevada, Oklahoma, Tennessee, and Wisconsin; from seven counties in Mississippi; eight in Missouri; 12 in Iowa; 16 in Illinois; 19 in Louisiana; and 32 in Texas. These figures definitely show an increase in the number of new anthrax areas in the past seven years.

The source of infection in most outbreaks occurring in new areas was not determined but in many instances contaminated food was suspected. It is interesting to note that a number of reports in foreign literature have appeared incriminating animal proteins and fodder as the source of infection.

From 1945 through 1951, the following 13 states reported no disease: Arizona, Connecticut, Delaware, Georgia, Idaho, Maine, Michigan, Montana, New Hampshire, Rhode Island,

TABLE 3. States Reporting Anthrax Outbreaks in Livestock* from 1945-1951 Inclusive, and Data on Incidence of

TABLE 3. States Reporting Anthrax Outbreaks in Livestock* from 1945-1951 Inclusive, and Data on Incidence of the Disease

	1945		1946		1947		1948		1949		1950		1951		Total	
	Number outbreaks	Livestock losses	Number outbreaks	Livestock losses	Number outbreaks	Livestock losses	Number outbreaks	Livestock losses	Number outbreaks	Livestock losses	Number outbreaks	Livestock losses	Number outbreaks	Livestock losses	Number outbreaks	Livestock losses
Alabama	1	3	1	1	0	0	2	10	1	1	2	6	1	2	8	23
Arkansas	0	0	0	0	20	60	0	0	1	1	0	0	4	8	25	69
California	47	170	38	239	31	259	28	149	25	315	14	83	18	201	201	1416
Colorado	1	8	2	45	0	0	0	0	0	0	0	0	1	5	4	58
Florida	0	0	0	0	0	0	0	0	0	0	0	0	21	209	21	209
Illinois	0	0	2	15	1	1	1	1	3	6	0	0	14	287	21	310
Indiana	0	0	0	0	0	0	0	0	1	7	0	0	1	4	2	11
Iowa	0	0	1	1	0	0	0	0	0	0	1	4	16	289	18	294
Kansas	1	5	0	0	0	0	0	0	0	0	0	0	0	0	1	5
Kentucky	0	0	0	0	4	35	0	0	0	0	0	0	12	193	16	228
Louisiana	0	0	1**	3000	0	0	1**	1200	18	298***	13	342	15	82	48	4922
Maryland	0	0	1	1	0	0	0	0	0	0	0	0	0	0	1	1
Massachusetts	0	0	0	0	1	7	0	0	0	0	0	0	0	0	1	7
Minnesota	10	13	0	0	1	3	3	13	1	1	0	0	3	17	18	47
Mississippi	7	35	9	66	4	52	30	118***	1	1	3	10	2	3	56	285
Missouri	3	30	48	239	24	113	9	15***	2	6	1	1	150	694	237	1098
Nebraska	3	34	3	15	4	15	11	17	7	12	7	7	3	7	38	107
Nevada	1	6	2	12	2	26	4	19	0	0	0	0	30	155	39	218
New Jersey	0	0	0	0	0	0	1	1	3	14	0	0	4	15	8	30
New Mexico	1	14	1	21	0	0	0	0	0	0	0	0	0	0	2	35
New York	1	1	3	3	4	4	5	8	4	6	2	3	1	1	20	26
North Carolina	0	0	1	1	0	0	0	0	0	0	0	0	0	0	1	1
North Dakota	2	28	0	0	0	0	0	0	0	0	0	0	1	5	3	33
Ohio	0	0	0	0	0	0	0	0	0	0	0	0	1	50	1	50
Oklahoma	0	0	0	0	1	28	0	0	0	0	0	0	5	39	6	67
Oregon	0	0	0	0	1	6	0	0	0	0	1	1	5	14	7	21
Pennsylvania	0	0	1	2	1	3***	0	0	1	8***	0	0	0	0	3	13
South Dakota	12	32	29	80	8	30	11	30***	11	15***	7	8	7	31	85	226
Tennessee	0	0	0	0	0	0	0	0	0	0	0	0	140	265	140	265
Texas	7	204	19	277	14	233	13	44	13	81***	9	129	25	172	100	1140
Utah	0	0	1	1	0	0	0	0	0	0	0	0	0	0	1	1
Virginia	0	0	0	0	1	3	0	0	0	0	0	0	0	0	1	3
Washington	0	0	0	0	1	1	0	0	0	0	1	1	0	0	2	2
Wisconsin	0	0	0	0	1	1	0	0	1	1	0	0	3	5	5	7
Wyoming	0	0	0	0	0	0	1	29	0	0	0	0	0	0	1	29
Total	97	583	163	4019	124	880	120	1654	93	773	61	595	483	2753	1141	11257

*Cattle, horses, sheep, and hogs.
**Major outbreaks involving numerous premises.
***Losses estimated.

South Carolina, Vermont, and West Virginia.

During this period, 29 cases of agricultural anthrax in man acquired by direct contact with the carcasses of animals dead of the disease were reported to the Bureau. Twenty-one cases were contracted by agricultural workers and eight by veterinarians. Records

TABLE 4. Summarized Data on Incidence of Anthrax in Livestock from 1945 to 1951 Inclusive

Year	No. states reporting	No. counties involved	No. outbreaks	Livestock losses*
1945	14	52	97	583
1946	18	82	163	4019**
1947	19	65	124	880
1948	14	63	120	1654**
1949	16	57	93	773
1950	12	55	61	595
1951	25	113	483	2753
Total			1141	11257

*In some states losses were estimated.
**Heavy losses were reported during severe epizootics of the disease in Louisiana.

of the U. S. Public Health Service from January 1, 1945 to December 29, 1951 list 379 cases in man, most of which were of industrial origin occurring principally in the New England states, New York, New Jersey, and Pennsylvania.

Records of the Federal Meat Inspection Division indicate that from July 1945 through December 1951, at establishments operating under federal meat inspection, 367 hogs and 39 cattle were condemned for anthrax (including spore vaccine lesions).

Summary and Conclusions

Results of a nation-wide survey to determine the incidence of anthrax in livestock in 1951 indicate that 483 outbreaks in livestock were reported from 25 states involving 113 counties with a total loss of 2,753 animals. No major outbreaks were reported. Of unusual interest is the large number of outbreaks of undetermined origin that occurred in swine in new areas. The total number of reported deaths in swine was 1,088 as compared with a total of 1,001 deaths in cattle. At establishments operating under federal meal inspection, 232 swine carcasses were condemned. Of the 483 outbreaks in 1951, 290 occurred in the states of Missouri and Tennessee with a total loss of 595 head. Outbreaks in new areas in 1951 were reported from 51 counties in 15 states. Outbreaks of the disease were reported in deer in Florida and in mink in Wisconsin. Seven cases of agricultural anthrax in man were reported to the Bureau; three of these were veterinarians.

During the seven-year period 1945 through 1951, there were 1,141 anthrax outbreaks in 35 states with losses of 11,257 head of livestock. Outbreaks in new areas were reported from 133 counties in 27 states. A greater number occurred in 1951 involving more counties than in any single year from 1945 through 1950. Twenty-nine cases of agricultural anthrax in man (1945 through 1951) were reported to the Bureau; 21 cases were agricultural workers and eight were veterinarians.

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Basic Code Covering Poultry Processing Sanitation

Basic proposals covering poultry processing sanitation prepared for adoption by cities and states, or to serve as a guide for local ordinances, are being prepared under the auspices of the Institute of American Poultry Industries.

The need for a code has been apparent for some time. Regulations adopted by various political divisions have in some instances acted as trade barriers. Some serve only as revenue measures and thus benefit no one. Public health representatives will serve with industry leaders to prepare a sanitary code which promises to assure consumers chickens safe to eat. The Institute is to be congratulated for its sincere efforts to establish uniform regulations which will govern operations of members of that group.

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No further evidence of foot-and-mouth disease has been reported in Mexico since the outbreak approximately one year ago. It is anticipated that the border will be open for importations as planned on September 1, 1952.

Production Of Enterotoxin By Staphylococci Recovered From The Bovine Mammary Gland

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THE role of milk and milk products in food poisoning, as well as the role of the Staphylococcus and its enterotoxin, has recently been reviewed by Dack[4]. The reader is referred to this publication for a complete review of the subject.

In some instances of staphylococcal food poisoning from milk or milk products it was evident that the organisms were shed in the milk from the mammary gland[1,8]. In a series of 160 cultures of staphylococci, isolated mainly from clinically normal mammary glands, one of the writers (Bell) found that 68% gave a positive test for gelatinase. It has been suggested that the ability to liquefy gelatin is indicative of the capacity of the staphylococci to elaborate the enterotoxin[9]. However, gelatinase production is not considered by some workers[2,5,9] a reliable means of distinguishing the enterotoxin-producing strains from those lacking this ability. It was desirable, therefore, to investigate further the toxin-producing ability of the staphylococcal cultures that had been isolated routinely from quarters with little or no visible evidence of infection. This report gives the results obtained when 37 of the cultures of staphylococci recovered from the bovine mammary gland were studied for enterotoxin production by means of the kitten test of Dolman and Wilson[7]. Results of the kitten test are correlated with the characteristics of pigment formation, hemolysis, coagulase production, and gelatinase production by the cultures.

Materials and Methods

Milk samples, collected under aseptic conditions, were cultured in ox-blood agar (blood agar base [Difco], plus 5% defibrinated ox-blood). At the end of 24 hours' incubation at 37.5 C. the presence or absence of hemolysis was noted and the colonies transferred to brain-veal or heart-infusion agar slants. Coagulase production was determined according to the method described by Thompson and Kharaza[10]. Gelatinase production was determined according to the method of Stone,[9] using either Stone's extract gelatin agar (Difco) or Staphylococcus medium no. 110 (Difco).

The method described by Dolman and Wilson[7] was the basis for the enterotoxin production and detection. Destruction of the toxic factors, other than the enterotoxin, was accomplished by heating the filtrates in a boiling water bath for 30 minutes. Following sterility checks, the filtrates were injected intraperitoneally into young kittens. Appearance of nausea, vomiting, and diarrhea, or profuse diarrhea with abdominal pain in a period of 15 minutes to two hours was considered to be evidence that the filtrate injected contained the enterotoxin. Filtrates prepared from uninoculated portions of the medium, handled in a manner similar to the inoculated portions, served as controls.

Results and Discussion

Thirty-seven cultures of staphyloccocci recovered from 37 different quarters of 27 cows were checked by means of the kitten test for the ability to produce enterotoxin. Twenty-five (67.5%) of the 37 cultures produced nausea, vomiting, and diarrhea, or profuse diarrhea with abdominal pain in a period of 15 minutes to two hours. No symptoms were produced by the control filtrates. The data, which supports that of Dolman, Wilson and Cockcroft,[6] and Dolman and Wilson,[7] indicate therefore that a high percentage of the cultures examined were able to produce the enterotoxin. The possibility exists, however, as Dolman and Wilson[7] point out, that the cultures examined would produce the enterotoxic factor only under the conditions of the experiment. The potential ability of a large number of cultures of staphylococci associated with the mammary gland to produce enterotoxin, on the other hand, emphasizes the need for adequate pasteurization of milk in order to eliminate this hazard. It would also

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seem desirable to give much consideration to the staphylococci in mastitis control programs, even though they may not be the most important organisms or even responsible for clinical symptoms.

The heat resistance of the enterotoxin in the filtrates prepared, making it possible to remove the other toxic factors by the heating procedure, is in agreement with the findings of Woolpert and Dack,[11] Dolman and Wilson,[7] and Dolman, Wilson and Cockcroft[6].

Comparisons of enterotoxin with pigmentation, hemolysis, and coagulase production, and with gelatinase production are shown in the following tables.

TABLE 1. Comparison of Pigmentation, Hemolysis and Coagulase Production with Exterotoxin Production

Hemolysis, pigmentation coagulase		Enterotoxin produced	
Result	No.	+	—
Positive	26	20	6
Negative	8	5	3
Intermediate*	3	0	3

*Positive for one or more but not all three of these characteristics.

It is seen from table 1 that a majority of the cultures that were hemolytic, orange-pigmented and coagulase-positive were capable of producing the enterotoxin. The possession of these characteristics, although indicative of enterotoxin producing ability, cannot, however, be relied upon always to distinguish the enterotoxin-producing and the non-enterotoxin-producing strains since it is also evident that the majority of the cultures negative for these characteristics also produced the enterotoxin.

TABLE 2. Comparison of Gelatinase Production with Enterotoxin Production

Gelatinase produced		Enterotoxin produced	
Result	No.	+	—
Positive	29	22	7
Negative	8	3	5

It is evident from table 2 that the ability to liquefy gelatin is not always associated with the ability to produce enterotoxin. There was agreement between these two characteristics in approximately three-fourths of the cultures examined under the conditions of the experiment. The fact that some gelatinase-positive cultures failed to produce the enterotoxin, while some gelatinase-negative cultures were capable of producing this toxin, makes it necessary to employ additional studies in order to determine the presence or absence of the capability.

Summary

Twenty-five of 37 cultures of staphylococci isolated from the bovine mammary gland produced enterotoxin under conditions of the experiment. Cultural and biochemical characteristics alone could not be relied upon to distinguish the enterotoxin-producing strains from those lacking this capability.

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Air Sac Infection in Poultry

A respiratory disease of poultry termed air sac infection has appeared in almost all major poultry producing sections of the country. Greatest losses have been suffered in the East. Mortality in some flocks has run as high as 30 to 50%.

Whether this is a new disease entity is questionable. A research project, or series of projects, have been proposed and are being set up in various parts of the country. Objectives are to determine the cause or causes, method of spread, and effective control measures.

Anamnestic Reaction As An Aid In The Differentiation Of Vaccinal From Infection Titers Of Brucellosis (Abortus) In Cows*

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WITH the advent of vaccination as a means for control of brucellosis in cattle, it has become a problem of importance to differentiate infection titers from vaccinal titers. Dick, Venzke, and York[1] reported on A Method for Differentiating Between Vaccinal Titers and Infection Titers of *Brucella abortus* in Cattle. Venzke[2,3] has made additional reports on this problem.

It is suggested that the basis for this test involves the anamnestic reaction (anamnesis-recollection). In this phenomenon there is a prompt and efficient production of antibodies following the secondary injection of a specific or nonspecific antigen. For a complete discussion of antibody production and the anamnestic reaction, the reader is referred to an article by Cannon[4].

Animals with a vaccinal titer will respond to an injection of *Br. abortus* strain 19 by a striking rise in the blood serum agglutinins within a maximum of 17 days[1,2,3]. This reaction in the case of noninfected animals may be called anamnestic, secondary immune response, or the reaction to stimulating, booster, or repeat injections. Diphtheria and tetanus toxoids, and typhoid-paratyphoid vaccine as booster antigens are now being used in human medicine and are examples of specific anamnesis[4]. Stimulating injections of *Br. abortus* strain 19 may be of value in the problem of brucellosis control.

Technic

Blood serum agglutination tests were made, using the rapid plate method, on all test animals. Commercial antigen of uniform sensitivity was used, and the testing plate was allowed to incubate for ten minutes before observations were made. For best results with this test, blood samples should be taken at intervals of seven to ten days to determine if the blood serum level is in a static, increasing, or decreasing phase. However, due to factors beyond our control, this was not carried out at regular intervals in this experimental work. When a static or declining titer was determined, the animal was injected intramuscularly with 5 cc. of *Br. abortus* strain 19 vaccine. Animals with an increasing titer were not injected because it was thought that subsequent rises would not be of diagnostic significance. Injected animals were bled at various intervals after injection and the blood serum titers were determined.

Numerous attempts were made to isolate Brucella from the milk of all animals before and after vaccine injection.

At the outset of this work, whey agglutination titers were determined routinely on milk samples from each quarter of the udder at the same time the blood samples were tested. There were so many variations and such an apparent lack of correlation that this phase of the problem was not thought to be of significance, and was discontinued.

Results

Results of the tests on a group of cattle vaccinated as calves and from which *Br. abortus* had been isolated previous to this test are shown in table 1. The prerevaccinal titers among these animals varied from 1:100 to 1:1600. With the exception of animal no. 128A, there was no rise of titer following the injection of strain 19. This animal is illustrative of the point that the three prerevaccinal titers should be in a static or decreasing titer phase. In an infected animal, the Brucella are continuously present as a stimulus to antibody production, and an additional stimulus by the

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injection of 5 cc. of *Br. abortus* strain 19 does not affect the titer of such an animal for approximately 16 days. A rise of titer from 1:100 to 1:400 was found 30 days after revaccination of animal no. 386A, indicating too long an interval.

Data are presented in table 2 showing the effect of strain 19 revaccination on cattle having a negative (1:50 dilution) blood titer. A high titer in approximately eight to 16 days was noted in each instance.

The effect of *Br. abortus* strain 19 revaccination on cows with a titer of 1:50 or higher is shown in table 3. There was a rise in titer in all animals of this group, with the exception of animals no. 136A and 354A, which exhibited prerevaccinal and postrevaccinal titers of 1:200 and 1:400, respectively. Since these two animals did not show a rise in titer, they probably should be considered as being in the same group as those animals represented in table 1, which is the control group of known infected animals. The remainder of the animals in this group should be considered to be in the same category as those listed in table 2.

Results of the test when applied to a group of cattle revaccinated as adults are shown in table 4. These cattle were negative when previously adult-vaccinated. The prerevaccinal titers varied from 0 to 1:200. In each instance there was a rise of titer and, therefore, such animals were adjudged to be noninfected. When negative animals which have not been vaccinated, or having been vaccinated and their respective titers having receded to less than 1:50, show a titer of 1:100 or higher subsequent to vaccination, they are classified as reactors. However, animals vaccinated as older calves or as adults frequently carry a variable titer (tables 3 and 4) over an indefinite period of time. These animals upon revaccination demonstrate a typical anamnestic reaction and, therefore, should be considered as noninfected reactors.

In table 5 the data are shown for two animals which were vaccinated as infected adults. The results of this test show these animals as being chronically infected.

All cows from which *Br. abortus* was isolated from the milk are listed in tables 1 and 5. Initial isolations were made before revaccina-

TABLE 1. Effect of Strain 19 Br. abortus Revaccination of Infected Cows*
(Br. abortus isolated from all animals previous to revaccination)
(Vaccinated as noninfected calves)

Cow No.	Age Yr.-Mo.	Prerevaccinal Titers		Titer Date Revaccinated	Postrevaccinal Titers		
124A	4-10	4-2-48 1:1600	4-16-48 1:1600	5-5-48 1:1600	5-14-48 1:1600	5-21-48 1:1600	6-4-48 1:1600
128A**	5- 0	12-6-48 1:400	12-16-48 1:400	1-20-49 1:1600	2-2-49 1:1600	2-11-49 1:1600	2-18-49 1:1600
129A	4- 2	4-1-48 1:200	4-16-48 1:200	5-5-48 1:200	5-14-48 1:200	5-21-48 1:200	5-31-48 1:200
133A	3- 8	3-10-48 1:400	4-1-48 1:400	5-5-48 1:400	5-14-48 1:400	5-21-48 1:400	5-31-48 1:400
139A	4- 0	12-6-48 1:800	12-16-48 1:800	1-20-49 1:800	2-2-49 1:800	2-11-49 1:800	2-18-49 1:800
152A	2- 8	12-6-48 1:1600	12-16-48 1:1600	1-20-49 1:1600	2-2-49 1:1600	2-11-49 1:1600	2-18-49 1:1600
372A	4- 5	12-6-48 1:400	12-16-48 1:400	1-20-49 1:400	2-5-49 1:400	2-11-49 1:400	2-18-49 1:400
386A	3- 4	3-10-48 1:100	4-1-48 1:100	5-5-48 1:100	5-14-48 1:100	5-21-48 1:100	6-4-48 1:400
388A	3- 2	4-1-48 1:400	4-13-48 1:400	5-5-48 1:400	5-14-48 1:400	5-21-48 1:400	5-31-48 1:400
391A	3- 3	9-10-48 1:1600	9-24-48 1:1600	10-1-48 1:1600	10-9-48 1:1600	10-14-48 1:1600	10-26-48 1:1600
392A	3- 6	12-6-48 1:1600	12-16-48 1:1600	1-20-49 1:1600	2-2-49 1:1600	2-11-49 1:1600	2-18-49 1:1600
465A	3- 9	3-10-48 1:1600	4-1-48 1:1600	5-5-48 1:1600	5-14-48 1:1600	5-21-48 1:1600	6-4-48 1:1600

*Selected group of infected cows wherein immunity was not established following calfhood vaccination with strain 19 *Br. abortus* vaccine.

**Brucella organisms were isolated from the milk of this cow 1-15-49 for the first time. In the case of all others cows, Brucella organisms were isolated from the milk before the date of the first prerevaccinal titer.

TABLE 2. Effect of Strain 19 Br. abortus Revaccination of Noninfected Cows
(Blood titer negative — 1:50 dilution)
(Vaccinated as noninfected calves)

Cow No.	Age Yr.-Mo.	Prerevaccinal Titers		Titer Date Revaccinated		Postrevaccinal Titers*		
127A	8- 3	12-6-48	12-16-48	1-20-49	2-2-49	2-11-49	2-18-49	5-5-49
		0	0	0	1:400	1:400	1:400	1:100
131A	4- 3	9-10-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	
		0	0	0	1:400	1:800	Sold	Sold
134A	4- 4	9-16-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	5-5-49
		0	0	0	1:400	1:400	1:800	1:100
249A	5- 9	9-16-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	5-4-49
		0	0	0	1:400	1:800	1:400	1:100
251A	4- 0	9-10-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	5-4-49
		0	0	0	1:200	1:400	1:100	1:50
255A	4- 3	9-10-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	5-9-49
		0	0	0	1:50	1:800	1:800	1:200
359A	5- 0	9-10-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	5-9-49
		0	0	0	1:1600	1:1600	1:400	1:50
457A	5-10	9-16-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	
		0	0	0	1:200	1:200	1:100	Sold
459A	5- 1	9-16-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	5-4-49
		0	0	0	1:800	1:800	1:800	1:100

*The fourth column is given for information as to the subsequent status of titer.

tion. Attempts were made repeatedly before and after revaccination to isolate *Br. abortus* from the udders of all cows listed in this study, but, except from those listed in tables 1 and 5, no isolations were made.

Summary and Conclusions

1. Twelve cows proved to be infected, by isolation of *Br. abortus* organisms from each, were vaccinated with strain 19 *Br. abortus* vaccine. Prevaccinal titers of the cows ranged from 1:100 to 1:1600. Only two of these cows showed a rise in titer after vaccination; the increase in titer of one probably being caused by lack of a static titer before vaccination and in the other to the long time interval after vaccination.

2. Nine cows showing a negative reaction

TABLE 3. Effect of Strain 19 Br. abortus Revaccination of Cows with Suspicious Titer or Higher
(Attempted isolation of Brucella not successful)
(Vaccinated as noninfected calves)

Cow No.	Age Yr.-Mo.	Prerevaccinal Titers		Titer Date Revaccinated		Postrevaccinal Titers*		
136A	3- 8	3-17-48	4-1-48	5-13-48	5-21-48	5-31-48	6-4-48	
		1:400	1:400	1:400	1:400	1:400	1:400	Sold
244A	4-11	9-16-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	5-10-49
		1:50	1:50	1:50	1:400	1:800	1:800	1:100
248A	5- 0	12-6-48	12-16-48	1-20-49	2-2-49	2-11-49	2-18-49	5-4-49
		1:100	1:100	1:100	1:800	1:800	1:800	1:200
256A	3-10	9-16-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	5-4-49
		1:50	1:50	1:50	1:200	1:800	1:1600	1:400
264A	2- 6	12-6-48	12-16-48	1-20-49	2-2-49	2-11-49	2-18-49	5-4-49
		1:200	1:200	1:200	1:400	1:400	1:400	1:200
265A	2- 6	12-6-48	12-16-48	1-20-49	2-2-49	2-11-49	2-18-49	5-4-49
		1:100	1:100	1:100	1:800	1:800	1:800	1:200
354A	5- 6	3-10-48	3-16-48	5-5-48	5-14-48	5-21-48	5-31-48	5-5-49
		1:200	1:200	1:200	1:200	1:200	1:200	1:50
380A	4- 4	9-16-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	
		1:50	1:50	1:50	1:1600	1:1600	Sold	Sold
395A	3- 2	9-10-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	5-4-49
		0	1:50	1:50	1:400	1:800	1:400	1:50

*The fourth column is given for information as to the subsequent status of titer.

TABLE 4. Effect of Strain 19 Br. abortus Revaccination of Adult Cows
(Attempted isolation of Brucella not successful)
(Vaccinated as nonreacting adult cows)

Cow No.	Age Yr.-Mo.	Prerevaccinal Titers		Titer Date Revaccinated	Postrevaccinal Titers*			
114A	5- 8	4-9-48	4-16-48	5-5-48	5-14-48	5-21-48	5-31-48	5-5-49
		0	0	0	1:200	1:200	1:200	1:50
194	8- 0	3-10-48	4-1-48	5-11-48	5-21-48	5-31-48	6-4-48	5-5-49
		1:100	1:50	1:50	1:1600	1:1600	1:1600	1:200
200A	9-10	3-10-48	4-1-48	5-11-48	5-21-48	5-31-48	6-4-48	5-5-49
		1:200	1:100	1:100	1:1600	1:1600	1:1600	1:200
230A	6- 5	3-10-48	4-1-48	5-5-48	5-14-48	5-21-48	5-31-48	
		1:200	1:100	1:100	1:200	1:800	1:1600	Sold
233A	5- 7	3-9-48	4-1-48	5-11-48	5-21-48	5-31-48	6-4-48	5-4-49
		1:100	1:100	1:100	1:200	1:400	1:800	1:200
328B	9- 6	9-10-48	9-27-48	10-1-48	10-9-48	10-16-48	10-26-48	5-5-49
		1:50	1:50	1:50	1:400	1:400	1:1600	1:50
347A	6-10	9-16-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	
		1:200	1:200	1:200	1:200	1:400	1:400	Sold
365A	5-11	3-17-48	4-1-48	5-5-48	5-14-48	5-21-48	6-4-48	10-1-48
		0	0	0	1:100	1:100	1:100	1:50
366A	5- 6	12-6-48	12-16-48	1-20-49	2-2-49	2-11-49	2-18-49	5-4-49
		1:100	1:100	1:100	1:400	1:800	1:400	1:100
371A	5- 7	9-10-48	9-25-48	10-1-48	10-9-48	10-16-48	10-26-48	
		1:100	1:100	1:100	1:200	1:200	1:200	Sold

*The fourth column is given for information as to the subsequent status of titer.

were likewise vaccinated. All of these cows showed a high titer in from eight to 16 days.

3. Nine cows classified as suspicious were revaccinated and all animals showed a marked rise in titer, except two. These two had prerevaccinal titers of 1:200 and 1:400, and it may be assumed that since they failed to show a rise in titer they should be classified as infected.

4. Ten cows which previously had been vaccinated as nonreacting adults were revaccinated with strain 19 *Br. abortus*. Agglutination reactions just previous to revaccination ranged from negative to 1:200. Without exception, the animals in this group showed a marked rise in titer after revaccination.

5. Two animals previously vaccinated as known infected adults were revaccinated. Both remained static in titer of 1:400 before and after revaccination.

6. With a few explainable exceptions, all revaccinated (a) nonreacting cows, (b) suspicious cows, and (c) previously adult-vaccinated, negative cows exhibiting varying reactions, showed a marked rise and subsequent decline in titer. Also, no elevation in titer resulted from the vaccination of (a) known infected animals, and (b) known infected animals previously adult-vaccinated as reactors. It would seem, therefore, that the anamnestic reaction is sufficiently reliable to be of value as a diagnostic measure in differentiating between vaccinal titers and infection titers.

Acknowledgment

The authors express thanks to members of the Department of Bacteriology, Kansas State College, for their sincere cooperation.

(Continued on page 348)

TABLE 5. Effect of Strain 19 Br. abortus Revaccination of Infected Cows
(Br. abortus isolated from all cattle previous to revaccination)
(Vaccinated as infected cows)

Cow No.	Age Yr.-Mo.	Prerevaccinal Titers		Revaccinated Titer Date	Postrevaccinal Titers		
219A	7-10	4-1-48	4-16-48	5-5-48	5-14-48	5-21-48	5-31-48
		1:400	1:400	1:400	1:400	1:400	1:400
450A	5- 8	4-1-48	4-16-48	5-5-48	5-14-48	5-21-48	5-31-48
		1:400	1:400	1:400	1:400	1:400	1:400

Treatment Of Feline Infectious Enteritis In A Cat Colony With Antiserum And Aureomycin*

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FELINE infectious enteritis, or acute infectious feline panleukopenia, was accidentally introduced into a laboratory colony of 61 cats of various ages. Four months prior to the outbreak, 32 of the 56 cats in the colony at that time had been immunized against feline infectious enteritis. Of these 32 cats, nine received their first immunization consisting of two doses of 2 cc. each, administered subcutaneously, of a commercial vaccine prepared from infected kitten tissues in which the virus had been inactivated with formalin. The remaining 23 had had one or more previous courses of injections at yearly intervals.

All adult animals were housed in individual cages in three separated rooms, but both immunized and unimmunized animals were kept in each of the rooms. There had been no known previous outbreaks of feline infectious enteritis in the colony since its establishment four years earlier, and at the time of this epizootic, all cats had been in good health with one exception. Approximately 14 days before the outbreak, unimmunized cat no. 1 exhibited respiratory symptoms which became increasingly severe, characterized by a clear, thin, mucoid nasal discharge, sneezing, and labored respirations. The following day, or 13 days before the outbreak, two apparently normal kittens, with unknown histories, animals no. 25 and 26, were introduced into the colony and housed in the same cage with a slightly older, unimmunized kitten, no. 17. Three more kittens, animals no. 27, 28, and 29, were added five days later and were housed together in one cage. These two cages were in the same room with cats no. 1, 4, and 5 and two actively immunized cats. Animals no. 25 and 26 died without showing obvious symptoms five days before the general outbreak. On the day before the outbreak, animal no. 27 died, and animals no. 28 and 29 were noted to be sick. These latter two died the following day, at which time the entire colony was examined for symptoms of infectious enteritis, and treatment started.

The 32 actively immunized cats were given no treatment at any time. There was no evidence of feline infectious enteritis among the animals in this group, although they were exposed to the infection. All ate well and remained normal throughout the course of the outbreak.

Treatment

For purposes of treatment, the unimmunized cats were divided into two groups: group I, three cats showing symptoms of feline infectious enteritis, including cat no. 1 which had been suffering from severe respiratory infection; group II, 21 cats without observable symptoms. Details of the outbreak are presented in table 1.

*Observations reported in this paper were made during the course of experiments on reproductive and maternal behavior in the domestic cat, supported by a grant made to Dr. Lester R. Aronson, by the Committee for Research in Problems of Sex, National Research Council.

†Department of Animal Behavior, American Museum of Natural History; Public Health Service Research Fellow of the National Institute of Mental Health.

TABLE 1

Group No.	Cat No.	Wt. Lb.	Temperatures Dec. 19	20	21	22	23	24	25	Antiserum cc./lb. Dec. 19	20	21	22	Aureomycin 50 mg./dose No. doses 19	20	21	22	White Blood Count Dec. 19	20	22	Jan. 2	Comments
I	1	4.55	6.0	6.5	4.8	2.8				2.0				1	2	2		1,500		6,750	15,500	Resp. symp. 14 days before outbreak.
	2	2.6	2.0	3.0						2.0				1	1				1,500			Died 12.20.
	3	3.0	5.5	4.6	2.0					2.0				1	2	1						
II	4	5.61			4.5	5.0				1.0		1.5				2	2			2,150		Died 12.25.
	5	8.99			3.0	2.8				1.0						2	2					
	6	2.8	3.0	5.2	5.0	3.4	2.8	—	2.6	1.0			1.0		2	2				3,400		
	7	2.8		2.5	3.0					1.0					2	1	1			1,500	21,650	
	8	4.6		5.0	4.4	4.4	2.2			1.0			1.0		2	2						
	9	4.6		5.0	5.0	5.0	3.2			1.0			1.0		2	2						
	10	2.6		2.4	1.4					1.0					1	1	2					
	11	4.53		4.2	3.6					1.0					1	2	1					
	12	2.95		1.4						1.0												
	13	6.77		2.4	2.5					1.0					1	1	2					
	14	4.5		3.0	2.2					1.0					2	1*	1					
	15	1.8		4.0	2.4	2.6	1.2			1.0					2	1	1					
	16	1.9		2.0	2.2					1.0					2	1	1					
	17	2.0		3.0	2.0					1.0					2	1	1					
	18	1.9		3.0	1.4					1.0					2	1	1					
	19	2.0		2.4	2.3					1.0					2	1	1					
	20	2.7		2.0						1.0			1.0									
	21	5.81		3.5	2.5	3.0	(1/2-3.4)			1.0	1.0		1.0		2	1	1					
Kittens of #21			12/27		12/28		1/2															
	22	1.12	1.6		2.4		2.0			1.0												
	23	1.0	2.8		2.4		2.0			1.0												
	24	1.28	5.0		2.4		0.0			1.0												

Severe symptoms feline infectious enteritis: lost 156 gm. May not have received full serum dosage.

In addition to the characteristic visible symptoms in the animals in group I at the start of treatment, there were elevated temperatures, 105.5 F. and 106 F., in cats no. 1 and 3, and a white blood count of 1,500 in cat no. 1. On the second treatment day, a similar blood count was obtained for cat no. 2. The animals in this group each received one dose of feline distemper antiserum at the rate of 2 cc. per lb. of body weight, subcutaneously, and 50 mg. (approximately 12.5-25 mg./lb.) of aureomycin orally on the first day. Aureomycin treatment was continued on the second and third days.

To each of the 21 cats in group II, one dose of antiserum was administered at a dosage of 1 cc. per lb. of body weight. Aureomycin therapy was instituted in the majority of cats in this group on the second day because of loss of appetite, elevated temperatures, and in some cases, diarrhea. Antiserum†† and aureomycin†† were repeated in these animals as indicated by the condition of the individual. However, when once started, it was generally planned to administer the antibiotic every 12 hours until four doses had been given.

TABLE 2

Kitten No.	6/8	6/9	6/10	6/11	6/12	6/13	6/14	6/15	6/16	6/17	6/18	6/19	6/20	6/21
1	N	N	N	2,400	H									
2	N	N	N	N	N	N	N	N	N	N	N	N	N	N
3	N	N	N	N	N	Dead								
4	N	N	N	N	N	N	1,900	H						
5	N	N	N	N	N	N	N	N	N	N	1,600	H		
6	N	N	N	N	N	N	N	N	5,900	3,800	1,400	H		
7	N	N	Dead											
8	N	N	N	6,800	3,900	Dead								
9	N	N	N	N	N	2,000	H							
10	N	N	N	N	N	N	N	N	N	N	1,500	H		
11	N	N	N	1,400	H									
12	N	N	Dead											

N—normal white blood count
H—tissues harvested

Results

Cat no. 2, three months of age, extremely sick and too weak to stand when treatment was started, died on the second day. Respiratory symptoms, probably unrelated to feline infectious enteritis, completely disappeared in cat no. 1, 24 hours following initial treatment. Uneventful recovery followed in this animal and cat no. 3 in four and three days, respectively.

There was one death in group II. This animal had received injections of feline distemper vaccine eight days and one day before administration of a single dose of antiserum. It showed no symptoms of feline infectious enteritis until two days after initial treatment, at which time aureomycin therapy was started, but no further antiserum was given. The animal died on the fifth day of illness. The remaining 20 cats in this group showed complete clinical recovery in three to five days.

As indicated in the table, white blood counts rapidly returned to normal.

In retrospect, it is apparent that cat 21, the nursing mother of three 45-day-old kittens, animals 22, 23, and 24, should have been included in group I for the purpose of treatment. It was noted that she did not eat well the day before treatment was started, but her general behavior was such that this factor was discounted. Subsequently, she became extremely ill, but eventually recovered. Of her kittens, animals 22 and 23 never developed any symptoms, and, despite the sudden weaning necessitated by the mother's condition, lost no weight. Animal 24, which had started eating solid food just prior to the outbreak, developed severe symptoms and lost 156 gm. before recovery. At the time of treatment, it was considered possible that this kitten might not have received a full prophylactic dose of antiserum.

Comment

Results obtained in this outbreak are essentially in agreement with results reported by

††Feline distemper vaccine, feline distemper antiserum and aureomycin were kindly furnished by Lederle Laboratories Division, Pearl River, N. Y.

others. Hage[1] stated that, while response to treatment was dependent upon the virulence of the virus, he has experienced only a 5 to 10% mortality in cats treated with aureomycin, while 90 to 95% mortality is expected in untreated cases. He recommended dosage at the rate of 50 mg. orally, at 12-hour intervals for three to four days, and stated that, in his experience, results following the use of aureomycin alone were as good as those following the use of serum and aureomycin. Slappey and Stapleton,[2] using one dose of 5 cc. of feline antibacterial serum, plus 50 mg. of aureomycin repeated every 12 hours for a total of five to eight doses, in 80 cats, reported a recovery rate of 85% instead of the fatality rate of 85% previously experienced.

While there is no evidence of a direct effect of aureomycin on the virus of infectious enteritis in controlled laboratory experiments, excellent response has been obtained in naturally infected animals. Its ease of administration, quick clinical response, and low toxicity suggest its value in clinical use in feline practice.

ADDENDUM

Two additional outbreaks of panleukopenia have occurred approximately six months following the outbreak reported. These incidents are of interest for the following reasons:

1. They confirm the effectiveness of the distemper vaccine in protecting cats and kittens from the disease.

2. They confirm our previous observations that, in our experience, a combination of antiserum and aureomycin can bring about rapid clinical recovery even when treatment is started at a late stage.

3. They lend support to the belief that, even though there are no active cases in the area, the virus remains virulent for long periods and unprotected cats may contract the disease.

Outbreak No. 1

On May 2, 1951, there were approximately 60 immunized adult cats and kittens in the laboratory. Eight apparently normal kittens, six to eight weeks of age, were introduced into the colony, and each received the first 2 cc. immunizing dose of feline distemper vaccine. One week later, on May 8, kittens no. 30 and 31, littermates housed in the same cage, were extremely ill. Kitten 32, also housed in this cage, remained healthy throughout the epizootic. In an adjacent cage in the same room, kitten 33 appeared sick, and its cagemate, kitten no. 34, remained healthy. In a third cage in this room, kittens 35, 36 and 37, all remained well. Other rooms in the laboratory at this time housed five adult unimmunized cats and about a dozen unimmunized nursing kittens. None of the above unprotected adults showed any symptoms, nor did any of the protected cats show any symptoms.

Kitten 30 died before treatment was instituted. Autopsy revealed lesions typical of panleukopenia. Kitten 31 was comatose, with occasional convulsions and vomiting, temperature of 99 F., very rapid and weak pulse, and a white blood count of 7,000. This kitten received 50 mg. aureomycin orally, and feline distemper antiserum at a dosage of 2 cc. per lb., subcutaneously. On the following day, one 50 mg. dose and one 25 mg. dose of aureomycin were administered. This kitten was already showing remarkably rapid recovery at this time. Clinical recovery was complete on May 11, the fourth day from the start of treatment.

The unprotected adults, and the older nursing kittens which had been in the colony were all given doses of 1 cc. antiserum per lb. of body weight and 25 mg. aureomycin per animal. Kittens 32-37 were treated in the same manner initially, and aureomycin treatment was repeated on the second day. Clinical recovery was apparent on the third day in kitten 33, which was showing symptoms. All other unprotected animals apparently remained normal.

Outbreak No. 2

About two weeks later, nine apparently healthy kittens were brought into the colony. Within 48 hours, three of these were disposed of and no further records on them are available. Two of the remaining kittens died within four days, exhibiting clinical symptoms of infectious enteritis.

White blood counts of the four remaining kittens were made, with the following results: 1,600, 1,500, 1,100, and 5,700, respectively. On the following day, kittens 1, 2, and 3 were destroyed, and spleens and sections of liver of each were harvested aseptically. At this time, white blood count of kitten 4 was 2,100, and it was destroyed and spleen and liver harvested. A pool of spleens from these four animals was homogenized and injected into 12 supposedly susceptible kittens. White blood counts were made daily, and the condition of kittens is shown in table 2. Seven of the animals exhibited typical panleukopenia and were

(Continued on page 348)

Toxicology In Canine Medicine*

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TOXICOLOGY is that subdivision of the field of medicine which deals with the effects and detection of poisons, and includes methods of treatment of such effects with antidotes. There are many concepts but few satisfactory definitions of "poisons"; the simplest and best-understood is "too much." The reasons for the harmful reactions to too much of any product vary in accordance with the chemical and other effects on the body tissues. The work of Selye[27] on the general adaptation syndrome (GAS) and the alarm-reaction (A-R) as the sum of all non-specific phenomena elicited by sudden exposure to stimuli which affect the body, is helping to understand pharmacotoxicodynamics. Detoxication mechanisms are being considered in detail,[24] not only with respect to old drugs but more particularly with reference to new drugs being developed by research and submitted to the Federal Food and Drug Administration before release for the treatment of disease. These concepts will underlie and extend classifications of available, often empirical data with respect to the toxicity of various chemical products now set forth in the literature and in standard reference textbooks[1,2,3,6,7,8,10,13,14,15,16,17,21,22,23,24,25,28,29,30,35,37].

In a report two years ago[21] the toxicity of the common rodenticides was stressed, with method of treatment of dogs that might be poisoned by exposure to that type of poisons. In the preparation of this paper, an attempt has been made to expand the poisons considered, to include that group in canine therapeutics. Only a few references have been found in the literature, dealing specifically with injurious effects on dogs, in spite of the protests of certain groups that dogs are being poisoned and tortured routinely by scientific laboratories throughout the world. A studied effort has been made to collect and to evaluate all available published reports in this field; the data obtained have been consolidated in table 1. The figure reported in the numerator represents the consensus with respect to the fatal or killing dose; the figure in the denominator represents an opinion with respect to the therapeutic dose. Under particular conditions quantities quite different from these may apply. The literature permitted estimation of doses of 60 products, and incomplete or inconclusive information on many more. The need for definite efforts to obtain, record, and evaluate definite data on many more products on dogs seems to be one outstanding conclusion from this search.

One of the deficiencies in this field may be pointed out by the data in table 2. The values reported are taken from a report by Lehman[18] contrasting the acute and the chronic toxicity of the series of insecticides. The acute toxicity was reported following oral administration to rats; chronic toxicity was followed for single and for multiple applications to the skin of rabbits; also by determining the lowest concentration in the diet of rats and of dogs which produced gross effects. The original report records the principal pathological mischief produced. It will be noted that there is a rough agreement in the order of toxicity of these products following a single dose and following repeated exposure, but that the quantitative relationships vary. There is no constant relationship in the rabbit dermal doses after single and multiple exposure; also there is no constant relationship between concentrations in the diet of the rat and of the dog. The author concludes: "In general, it may be stated that dogs are more susceptible to injury from the insecticides than rats."

This becomes important when it is recognized that one of the chief sources of poisoning dogs has been secondary poisoning following the ingestion of rats dying from rodenticides. This has not been noted after red squill[19] but has been observed following thallium, 1080 or warfarin[20,31,33]. This is to be expected after direct contact or inhalation of many of the newly developed rodenticides[5,18].

Studies by Krantz and associates[11,12] have shown a peculiar species difference between dogs and other species of animals. In noting reactions to the administration of tween 20

*Presented at the 18th annual meeting of American Animal Hospital Association, Atlantic City, N. J., May 3, 1951.

†Professor of Pharmacology, Temple University Schools of Dentistry and of Pharmacy; president, Munch Research Laboratories, Inc., Upper Darby, Pennsylvania. Present address: medical director, Strong Cabb & Co., Inc., Cleveland, Ohio.

intravenously to dogs, an allergic response was observed, as shown by a prompt, transient fall in blood pressure, temporary recovery, then weakness, postural difficulties, scratching, generalized erythema of the skin and mucous membranes, salivation, nausea and vomiting. All dogs tended to recover from moderate doses (0.1 cc. of 5% solution, equivalent to 5 mg./kg.). Other tweens produced similar effects, and larger doses led to death. Similar effects and death developed in foxes, dogs, jackals, coyotes, and wolves were less powerfully harmed. This phenomenon was not observed after injections into men, raccoons, chickens or the common laboratory animals (cats, rabbits, guinea pigs, rats, and *Macacus rhesus* monkeys). Since antihistaminics such as pyribenzamine[21] or benadryl[22] prevented or relieved the symptoms, the liberation of histamine is believed to be the etiological factor in this allergic response by the dog family.

The evidence of possible danger from prolonged administration of various drugs is pointed out by Moskey[18]. Implantation of 15-mg. pellets of diethylstilbestrol in the neck of chickens and turkeys was effective as a tenderizing agent and fattening agent; but it has been alleged that feeding such heads to minks rendered them sterile. The possible danger from consumption of such food, as well as that from animals fattened by arsenical drugs or antibiotics needs and receives thorough study.

Why should current work on insecticides and rodenticides be considered in discussing canine toxicology? Because many rodents die from consuming poisoned baits. Due to their stability, substantial quantities of toxic residues are present in the carcasses which are readily consumed by dogs, causing poisoning and death. In the second place, many of the newer products may be aerosolized or used as fumigants, thus being inhaled by dogs. In the third place, a number of these insecticides have proved value for treatment of infestations of dogs themselves, being used as liquids, dusts, and shampoos; thus external application may be an important factor in damaging dogs. Ward[23] stresses the need to read the label on products such as dog shampoos with lindane, for example, and to follow directions thereon . . . a point which has been stressed frequently in other connections. These new products should be safe for use; if used as directed, and may be very dangerous if used otherwise. Pups are much more sensitive to the toxic action of

[21]Ciba Pharmaceutical Products, Inc., Summit, N. J.

[22]Parke, Davis & Company, Detroit, Mich.

TABLE 1. Estimated Fatal/Therapeutic Doses for Dogs (mg./kg.)

Product	Method of Administration		
	Oral	Subcutaneous	Intravenous
Aconitine	20/0.2	4/0.1	10/0.1
Acetanilide	700/100	—	—
Acetophenetidin	—/130	—	—
Aloe	—/2000	—/40	—
Amytal, sodium	125/20	—/100	75/45
Antipyrine	—/250	—	—
ANTU	25/—	—	—
Apomorphine HCl	—/2	—/0.1	—
Arecoline HBr	5/1.6	—	—/0.1
Arsenious trioxide	25/1	—/1	—
Neoarsphenamine	20/50	—	—
Aspirin	1000/100	—	—
Atropine sulfate	—/1	140/0.2	60/—
Barbital, sodium	500/150	—	—
Betanaphthol	100/20	—	—
Caffeine	140/25	110/—	—
Calciferol	5/0.5	—	—/0.25
Calomel	160/10	—	—
Camphor	500/150	—/750	—
Carbon tetrachloride	25 cc. /0.3 cc.	—	—
Chenopodium, oil	0.3 cc. /0.1 cc.	—	—
Chloral	3000/250	600/—	—
Chlordane	60/—	—	—
Cocaine HCl	—/3	80/2.5	25/—
Codeine	—/2	200/15	—
Copper sulfate	—/10	—	27/1
Cyanide, sodium HCN	3/1 2/1	5.36/— 3/—	—/0.25 1.6/—
DDT	750/—	—	75/—
DFP	—	—	3.4/—
Digitalis	—/10	—	100/10
Epinephrine	—	5/0.6	0.2/0.04
Ethanol	12.25/—	—	3.35/—
Ethyl aminobenzoate	—	—	400/—
Ethylene glycol	—/9 cc.	—/3 cc.	—
Fluoride, sodium	100/1	150/1	50/1
1080	0.10/—	—	—
Formaldehyde	—	350/—	70/—
Ipecac	—/250	1000/—	—
Isopropanol	6.15 cc./—	—	3.35 cc./—
Kerosene	30 cc./—	—	0.50 cc./—
Magnesium sulfate	6000/3000	1500/—	225/—
Mercuric chloride	60/2	15/1	4/—
Methylene blue	1000/—	—	125/40
Methyl salicylate	3 cc./—	—/2 cc.	3 cc./—
Morphine sulfate	400/8	400/8	400/8
Nicotine	1/—	Over 23/—	3/0.35
PABA	3000/—	—	—
Papaverine HCl	—	125/—	100/40
Parathion	5/—	—	—
Physostigmine	—/0.2	4/1	—
Picrotoxin	2.25/1	1.5/—	—/0.3
Pilocarpine	—/0.2	—	—
Potassium dichromate	40/2	—	—
Santonin	500/10	—	—
Scopolamine HBr	5/0.5	—	—
Sodium salicylate	—/100	—	1000/250
Strychnine	3/0.1	0.35/—	0.4/0.1
Thallium sulfate	15/8	—	—
Tween 20	—/1	—	—

lindane than are adult dogs; just as calves may die after exposure to concentrations of 0.05% whereas cows are unaffected by similar exposure to 0.3% solutions. The strength of dip or shampoo solutions, the preliminary treatment as well as after treatment, and the frequency of treatment are among the important factors associated with the safe use of these preparations.

The testimony of Woolley[86] before the House Committee on Agriculture on February 20, 1951, lists current insecticide requirements of the United States, in millions of pounds:

Copper chemicals	112
Benzene hexachloride	65
DDT	58
Calcium arsenate	52
Toxaphene	45
Lead arsenate	37
2-4-D	22
Dithiocarbamates	10
Rotenone	10

The list is extended to cover many other products for which the estimate exceeds a million pounds. The hazards for consideration here are related to frequent contact and to secondary poisoning in dogs, as well as to acute harm from a single exposure.

The concern of the federal government with such hazards for products for man or other animals was reflected in the passage of the Federal Food, Drug and Cosmetic Act; also more recently the Federal Insecticide, Fungicide and Rodenticide Act of 1947. Section 6 (a) (2) authorizes the Secretary of Agriculture to set up hearings to determine economic poisons, and quantities of substances contained in economic poisons, which are highly toxic to man. Regulation 162.8 states that when tested on the laboratory animals, mice, rats, and rabbits, highly toxic products produce death in half or more test animals after oral doses of 50 mg. or less/kg. in a single dose; 200 parts or less per million by inhalation; or 200 mg. or less/kg. following application to the bare skin of rabbits. Such products require special cautions and antidotes, also directions for use.

Regulation 162.116 (b) classifies such toxic materials as highly toxic; a second class is established having toxicities down to about one-tenth of the highly toxic class; a third group is still less toxic but requires some cautions, being about one-tenth as toxic as class two. The fourth class is comparatively free from danger. Cautions are required for members of the first three classes, to prevent injury to living man and other vertebrate animals.

TABLE 2. Acute and Chronic Toxicity of Certain Insecticides (Lehman,[13])

Product	Acute Toxicity	Chronic Toxicity			
		Dermal Applications Solutions, Rabbits		Feeding: Lowest Level Causing Gross Effects	
	Mean Lethal Dose Rats, Oral mg./kg.	Single mg./kg.	Multiple mg./kg.	Rats ppm./weeks	Dogs ppm./weeks
Parathion	3.5	50	5	25/4	—
Nicotine	10	50	40	60/43	—
Gamma benzene hexachloride	125	50	20	400/52	330/30
DDT	250	2820	150	100/104	2640/7
Chlordane	500	1880	40	250/12	660/4
Dichlordiphenyl-dichlorethane	2500	2820	100	2500/104	(1650/80 survived)
Methoxychlor	6000 plus	2820	600	5000/16	(10,000/26 survived)

Among the products listed in this regulation of interest in canine practice are acetone, ANTU, tartar emetic, arsenic compounds, BHC, carbon bisulfide, carbon tetrachloride, chlordane, creosote, cresol, cyanides, TDE, DDT, 2-4-D, fluorides, formaldehyde, HCl, hypochlorites, kerosene, mercurials, parathion, phenols, P, quaternary ammonium salts, 1080, strychnine, and toxaphene (chlorinated camphene).

Other classifications of toxicity have been proposed in the literature. Hodge and Sterner[9] propose six toxicity ratings: (1) extremely toxic single oral lethal dose of 1 mg. or less/kg.; (2) highly toxic between 1 and 50 mg./kg.; (3) moderately toxic, between 50 and 500 mg./kg.; (4) slightly toxic, between 0.5 and 5 gm./kg.; (5) practically non-toxic, between 5 and 15 gm./kg.; and (6) relatively harmless, dose of 15 gm. or more/kg. These

classifications roughly correspond, in probable lethal doses for man, to a taste; 1 grain, 1 teaspoonful; 1 ounce; 1 pint; 1 quart; and over 1 quart, respectively.

Carpenter, Smyth and Possani[4] have established a somewhat more detailed classification of 19 gradations; each level is one-half the previous concentration in air inhaled; for example, concentrations from 256,000 down to 16,000 parts per million are grouped as "slight". Air concentrations inhaled for four hours to produce toxic effects between 8,000 and 1,000 are "moderate"; between 500 and 62 as "definite"; and "serious" includes concentrations from 32 down to 1 ppm.

While these various classifications may differ somewhat in their standards, they are helpful in focusing attention on the toxicity of products ingested or inhaled or otherwise taken into the body. By comparing any of these criteria with the toxic and therapeutic values recorded in table 1, it becomes obvious that many substances administered to dogs, or accidentally ingested by them, fall in the class of highly or definitely toxic agents. Caution should attend the deliberate administration of such products; also characteristic symptoms associated with these toxic materials should be kept in mind in diagnosing sudden, suspicious symptoms in the practice of canine medicine. Such attention will permit more effective therapy with drugs, and more successful treatment of cases of poisoning. Our information is incomplete with respect to many of these substances; it will be materially strengthened if reports of canine toxicology are consistently reported in suitable scientific publications.

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(Continued on page 350)

Highlights Of The 89th A.V.M.A. Convention

THE Atlantic City meeting, the 89th in the history of the American Veterinary Medical Association, held June 23-26, 1952, was characterized by the large number of scientific papers, on a wide variety of subjects, which were presented, and the many conferences held before, during, and after the regular sessions. These, too, were devoted to a great number of activities for which the headquarters group of the A.V.M.A. furnished leadership. Atlantic City weather failed its billing; the early portion of the week a disappointment to the teenagers because of rain and cold that discouraged bathers, and trying to all during the latter portion due to the sudden heat wave in the East which included the convention city.

The Local Arrangements Committee's work was well planned by Dr. J. R. Porteus, Dr. R. P. Lawrence, and Dr. J. B. Engle. Preparations were detailed carefully and functioned efficiently to the credit of those who supported the officers of the General Committee.

Particularly commendable was the convention publicity handled by Mr. L. R. Fairall who arranged for press and radio releases for each day of the meeting and programs by air and radio designed to further good public relations.

On Saturday, June 28, 1952, the A.V.M.A. convention was featured on the National Farm and Home Hour broadcast. For the fourth consecutive year, the National Broadcasting Company has arranged for the program on a national network of stations, following the convention. This year Dr. W. L. Boyd, president, Dr. T. Childs, veterinary inspector general of Canada, Dr. B. T. Simms, chief of the BAI, and Dr. A. G. Madden, member, Committee on Public Relations, were presented to the radio audience.

The President's Address

In his presidential address, Dr. John R. Wells, West Palm Beach, Fla., called attention of his hearers to our rapidly increasing population. He referred to the lack of new land for food production and the importance of improving efficiency for food production and conservation of that we now produce. Veterinarians are in a strategic position to reduce losses now suffered in the livestock industry because of disease by the better application of the known scientific principles of modern veterinary medicine.

Pointing to the close relationship between certain problems of animal health and human health and the many diseases which are common to both animals and man, Doctor Wells proposed that the professions form a united front to collaborate on many of these problems. He recommended that the A.V.M.A. invite other medical professions to join in a national, interprofessional organization for meeting this need.

Doctor Wells also called attention to the threat of foreign diseases becoming established in America and stressed the importance of adequate staffs of trained veterinarians in the government service to serve in the role of guards against possible disease introduction from foreign lands.

Scientific Program

The importance of a satisfactory ration for calves was graphically illustrated in a discussion of the influence of proper rumen function on factors of growth and general health in these animals. Improved health has followed a high roughage feeding system supplemented by weekly rumen inoculations over that of calves supplied what has been considered highly nutritious concentrate feeds. The system provides (1) limited amounts of milk, (2) liberal feedings, from birth, of good quality alfalfa and grass hay, and (3) infusion of fresh rumen contents or inoculation with cud materials.

Failure to supply satisfactory amounts of suitable roughage appears to be the cause of formation of hair and fiber balls, according to the experiments reported by Dr. W. D. Pounden, Wooster, Ohio. The incidence of rough hair coats, "pot bellies," pneumonia, diarrhea, and coccidiosis, has been favorably influenced by the suggested feeding system in investigations conducted by the speaker.

In his discussion on some practical procedures in horse practice, Dr. W. F. Riley, Jr., East Lansing, Mich., referred to the efficacy of surital® sodium intravenously as an anesthetic agent. Addition of 1 cc. of curare per

®Parke, Davis & Company, Detroit, Mich.

President of the A.V.M.A.

Dr. W. L. Boyd

Following his graduation in 1909 from the Kansas City Veterinary College, Doctor Boyd joined the staff of that institution as instructor. In 1911, he resigned and accepted a position in the Division of Veterinary Medicine of the University of Minnesota. Since the establishment of the School of Veterinary Medicine at that institution, Doctor Boyd has functioned as director and administrative head.

Elected president-elect at the Milwaukee convention in 1951, Doctor Boyd automatically advanced to the presidency, succeeding Dr. John R. Wells, which position he will hold for the ensuing year.

100 lb. of body weight was followed by muscular relaxation and served to reduce the amount of agent required to effect surgical anesthesia. The combination is reported to have proved dependable. Little "paddling" and none of the violent movements are associated with recovery of consciousness.

Chloral hydrate with magnesium sulfate intravenously has a wide margin of safety as an equine hypnotic. Coupled with a suitable nerve block or infiltration with novocain, most surgical procedures can be accomplished without pain to the patient.

The tremendous expansion of artificial insemination of cattle, since the close of World War II, has forced a change in methods for those engaged in dairy cattle practice, according to Dr. Ryland Crashaw, Columbus, N. J. Nowadays the practitioner specializing in dairy practice must be a sterility expert. Dairymen are demanding this service of which they have become conscious following wider use of artificial insemination of their breeding herds.

Radiological localization of foreign bodies in the reticulum of cattle was the subject of discussion by Dr. Francis A. Spurrell and Dr. H. C. H. Kernkamp, both of the School of Veterinary Medicine, Minnesota University, St. Paul. The course taken by these bodies was shown to be varied. Localization makes possible accurate prognosis in individual cases.

Two important considerations were emphasized by Dr. W. E. LaGrange, Media, Pa., in regard to bovine leptospirosis. Urine of infected and convalescent cows is the prime source of infection, indicating the necessity for isolation of such cases. There is evidence to indicate that the disease exists in horses and is related to recurrent iridocyclitis.

Investigators at the Cornell Research Laboratory for Diseases of Dogs announced encouraging results in trials with a new "double-acting" vaccine prepared at that institution and designed to protect dogs against distemper and infectious hepatitis. According to Dr. James A. Baker, director, this new vaccine is safe and effective, providing durable immunity for animals in controlled experiments.

These researchers first developed a vaccine for infectious hepatitis obtained from infected dogs; then means for combining it with an avianized distemper vaccine. Supplies of the new vaccine are not yet available for general distribution.

Results following treatment of anaplasmosis-infected, splenectomized calves were reported by Dr. James G. Miller, Baton Rouge, La. Both aureomycin and terramycin inhibited growth of this blood parasite. A total of 32 infected calves treated survived, whereas 21 of 26 controls died.

Public response to a well planned educational campaign made possible reduction of the incidence of rabies in Buffalo, N. Y., from severe proportions to zero in the brief period of one year. Dr. R. B. McClelland cited the example to illustrate that means are at hand and when properly employed can effectively eradicate this centuries-old disease.

The practitioner, states Doctor McClelland, has three primary duties on a rabies control team. They are: (1) accurate clinical diagnosis; (2) protection of patients by immunization; and (3) education of his clients.

Start sutures ventral to the lower commissure of abdominal wounds in cattle to avoid herniation.—*A. E. McChesney, D.V.M.*

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So long as you are learning, you are not growing old. It's when a man stops learning that he begins to grow old.—*Hergesheimer.*

The New Officers

Brigadier General James A. McCallam, Washington, D. C., chief of the Army Veterinary Corps, was named president-elect. He will take office as the executive head of the Association in 1953. Dr. W. L. Boyd, St. Paul, Minn., assumed the presidency at the final general session and will serve as chief during the ensuing year. Vice-presidents selected were: Dr. Lawrence A. Mosher, Atlanta, Ga.; Dr. Mark L. Morris, Topeka, Kan.; Dr. Harold S. MacDonald, Toronto, Ontario, Canada; Dr. C. H. McElroy, Stillwater, Okla.; and Dr. Mason Weadon, Washington, D. C. Dr. Harry E. Kingman, Jr., Chicago, Ill., was named treasurer.

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Hog Cholera Vaccination and Vaccines

Two papers were presented on the subject of hog cholera vaccination and vaccines; one by Dr. H. E. Biester, Ames, Iowa, and a second, reporting returns of a survey, by Dr. W. A. Aitkens, Chicago, Ill.

New vaccines, introduced just one year ago, have emphasized the need of caution in vaccination procedures. Doctor Biester's report of experimental studies differs from logical conclusions developed from the field survey summarized by Doctor Aitkens. In the investigational work on animals that sickened after all methods of vaccination, hog cholera virus was incriminated. Results of the survey, consistent time interval between vaccination and illness, plus low mortality among those that sickened, suggest other causes for the postvaccination morbidity. Furthermore, difficulties were experienced, according to 151 practitioners reporting, in only 8.8% of herds vaccinated. In 91.2% of herds treated with the new products made available, no postvaccination illness was reported.

Considering the many and varied factors that may influence results in herds under all types of management, definite conclusions cannot be drawn. More experience is needed, as well as further studies to assess the influence of general health and management on the immunological response of swine.

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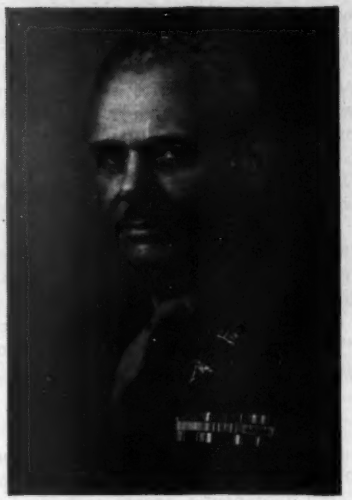
In order to fit a brucellosis testing and calfhood vaccination program into his practice, the veterinarian must plan and schedule necessary work carefully.—*A. M. Orum, D.V.M.*

President-elect of the A.V.M.A.



Brigadier General James A. McCallam

General McCallam was born in Philadelphia, Pa., on May 13, 1894. He received his veterinary degree from the University of Pennsylvania in 1917 and was appointed assistant veterinarian in the Veterinary Corps, Regular Army, with the rank of 2nd lieutenant on July 16, 1917. He graduated from the Army Veterinary School in 1924, and Army Medical Field Service School in 1925. He served with the Sixth Army in Australia, New Guinea, Leyte and Luzon in the Philippines, and finally as part of the occupation forces in Japan. General McCallam was awarded the Legion of Merit and the Bronze Star Medal for his outstanding service in the Southwest Pacific Theater.

The promotion of General McCallam to his present rank received Senate confirmation early in 1948, and his selection to head the Army Veterinary Corps was a fine compliment to the splendid service of this well qualified and popular officer and veterinarian.

General McCallam is the second military veterinarian and the first general officer to hold the office of president-elect since the founding of the A.V.M.A. in 1863. Colonel Robert J. Foster, V. C., U. S. A., retired, headed the Association in 1936-37.

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Toronto, Ontario, Canada in 1953

Convention dates for the 90th annual meeting were announced as July 20-23, 1953. Headquarters will be the Royal Hotel, one of the finest in Canada. These dates will allow ample time for travel for those who plan to attend the International Veterinary Congress to be held during August in Stockholm, Sweden. The House of Representatives accepted the invitation of the Minnesota group for Minneapolis in 1955. Seattle will be host in 1954.

Educational Exhibits

Twelve large educational exhibits furnished by institutions and other agencies, including the A.V.M.A., graphically portrayed a number of features of interest. These included anthrax in swine, method of handling fur animals, use of animals in medical research, pathology of total body radiation in dogs exposed to atomic explosion, leptospirosis in man and animals, bovine mastitis, teat injury, and meat inspection.

These exhibits devoted to modern problems in veterinary science are always educational. Qualified attendants, including veterinarians who have applied themselves to these specialties, were generally on hand to explain and discuss features presented.

Commercial Exhibits

A total of 62 commercial exhibits were set up in the headquarters hotel. These displayed the latest therapeutic agents and instruments designed to be useful for practitioners. Interest in these was intense and helpful suggestions were offered in new ideas. Visitors derived much benefit and technical assistance for the time spent discussing items on display with trained attendants who were anxious to be helpful.

Dr. Edwin Laitinen Elected Chairman of Executive Board

Dr. W. G. Brock, Dallas, Tex., who has served for the past year as chairman of the Executive Board of the A.V.M.A., was succeeded by Dr. Edwin Laitinen, practitioner, West Hartford, Conn.

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The threat of foreign disease introduction and spread of those diseases now present in this country by modern rapid transportation systems is grave. Dr. E. L. Stubbs, University of Pennsylvania, Philadelphia, pointed out that all methods of travel have produced situations that favor the passage of disease-producing agents without loss of virulence or deterioration. Fowl plague was singled out to illustrate the extent of disaster if this disease became established here. Referring to the 1924 outbreak in several eastern states, Doctor Stubbs recalled that losses during three trying months amounted to an estimated $1,000,000.

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A #15 Bard-Parker blade will cut through any horse's foot.—*W. F. Riley, D.V.M.*

The day has passed for the unqualified recommendation that lacerations in large animals should be treated as open wounds. Such wounds heal with excessive scar tissue. After suitable preparation, most all wounds can be sutured, provided modern methods and proper agents are employed. — *W. F. Riley, Jr., D.V.M.*

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Bovine brucellosis was named by the Committee on Diseases of Food-Producing Animals as the most serious disease of dairy and beef cattle. Mastitis is a close second among dairy cattle; anaplasmosis, shipping fever, and parasitism rank in order, as noted, in beef cattle.

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Dairy cattle are benefited by a 30-minute exercise period daily, regardless of weather conditions, during the stabling period.—*G. L. H. Weaver, D.V.M.*

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From a practical, as well as a pathological standpoint, I explain mastitis as a wound infection, producing little or large quantities of toxins, which in gangrenous mastitis result in a true case of blood poisoning.—*Robert Curtis, D.V.M.*

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Excess grain and too large quantities of milk appear to inhibit activity of essential rumen microorganisms and thus tend to interfere with proper growth and general health of young calves.—*W. D. Pounden, D.V.M.*

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Pigeons, turkeys, chickens, ducks, fulmars, gulls, parrots, and probably any bird can disseminate psittacosis — *David L. Coffin, D.V.M.*

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An increasing number of hospitals for the treatment and care of farm animals are being opened in rural sections in each state of the Union. This trend is favorable. Modern facilities provided assure patients the best care. Practitioners can employ available time more efficiently.

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Calfhood vaccination for brucellosis has increased significantly all over the nation, according to progress reports submitted by state livestock sanitary officials. Six states along the eastern seaboard now vaccinate 85% of all calves.

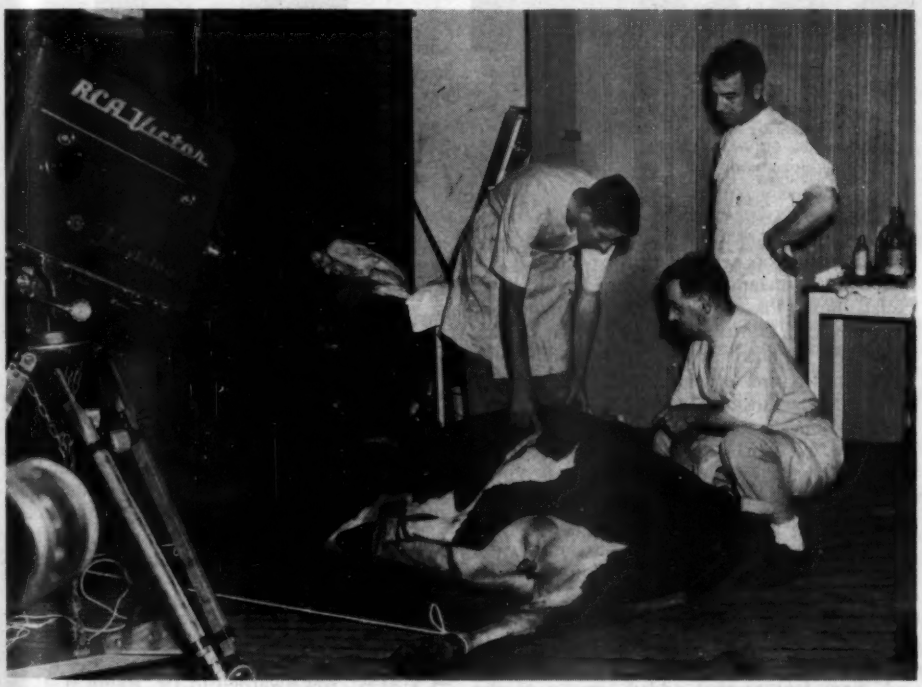
Television Program

Television portions of three section programs (General Practice, Small Animal, and Obstetrics and Surgery) highlighted each of these sectional meetings. There is no longer question of the value of this media (closed-circuit telecasts) at veterinary conventions. The manner of spacing small screens (40 individual receivers) along either side of the meeting room proved superior to the set-up last year. One could see and hear equally well from any seat in the auditorium. This portion of the scientific programs was sponsored by the Allied Laboratories, Inc. and the Radio Corporation of America (R. C. A.).

During the Monday afternoon Section on General Practice, Dr. J. V. McCohan, Downington, Pa., and Dr. J. A. Rogers, Bryn Mawr, Pa., demonstrated technic for intravenous medication for cows. This illustrated the use of the Kingma tube for slow infusion over extended periods. A new detail in the method was shown. This made use of a plastic container for parenteral solution which was adjusted over the back of the cow as a "saddle." Infusion tube was inserted into the mammary

Dr. Jacques Jenny (left), University of Pennsylvania, School of Veterinary Medicine, demonstrating application of the Schroeder-Thomas splint. Narrator (right) is Dr. J. A. S. Millar, practitioner, Deal, N. J.

Telecast of Bovine Cesarean Section



Standing (l-r) Dr. A. E. McChesney, Bozeman, Mont., who demonstrated technic used in his practice on range cattle. Dr. E. A. White (standing), East Lansing, Mich., who assisted Doctor McChesney, and Dean C. F. Clark (kneeling), East Lansing, Mich., who narrated the demonstration. Doctor McChesney advised his listeners and viewers that this operation is common in his practice because of range breeding of young and immature heifers.

vein. The method allows freedom for the patient and is satisfactory only with the animal in the standing position.

Dr. C. W. Raker, Philadelphia, Pa., demonstrated his technic for removal of the claw of a cow with a pruning (looping) shears. Dr. W. D. Pounden, Wooster, Ohio, demonstrated collection of rumen contents, examination of material collection, and transfusion to a calf. Here, to the delight of studio technicians and viewers, microscopic view of a fresh

Collection of rumen contents for examination and transfusion. Dr. W. D. Pounden, Wooster, Ohio, (1) demonstrated a practical method of obtaining rumen material by means of a stomach tube. He is assisted by Dr. A. G. Danks, Ithaca, N. Y., who also described the procedure.

slide of rumen contents was televised. Three other demonstrations were televised at the second session on general practice. Dr. A. D. Goldhalft, Vineland, N. J., Dr. Frank Kral, Philadelphia, Pa., and Dr. E. E. Chambers, Rossville, Ga., contributed. Dr. A. G. Danks, Ithaca, N. Y., acted as narrator for all.

Demonstrations of surgical technic by Dr. C. L. Blakely, Boston, Mass., Dr. W. F. Hoffman, Pittsburgh, Pa., Dr. L. R. Barto, Basking Ridge, N. J., Dr. Jacques Jenny, Philadelphia, Pa., and Dr. C. P. Zepp, Sr., and C. P. Zepp, Jr., were screened during the session on small animal practice.

Lighting, photography, and narration were near perfect in all of these, making possible close-up illustration of minute detail. Everyone seated in the large meeting room had a surgeon's-eye view of these operations. Only color needed to be added for perfect reality.

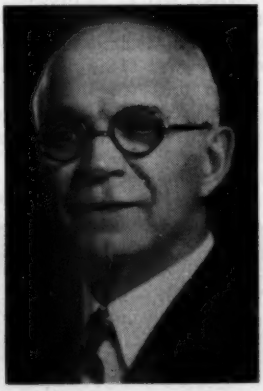
A final feature that added interest to the telecasting was provision for transmission of questions from the floor and their immediate answer by demonstrators in the studio.

The section on Surgery and Obstetrics included four surgical demonstrations during its two sessions.

Awards

Dr. Charles E. Cotton was awarded the highest honor the American Veterinary Medical Association can bestow on one of its members, the 12th International Veterinary Congress Prize.

Citation presented commended Doctor Cotton for his "forthright stand on disease control issues during his years of service as a livestock and sanitary official in Minnesota." Doctor Cotton started private practice in Minnesota in



Dr. Charles E. Cotton

1894, just one year after his graduation from the University of Pennsylvania. At the outbreak of World War I, he was among those instrumental in planning the Army Veterinary Corps as a branch of the armed services. He later served with that corps.

Doctor Cotton is a past president of the A.V.M.A., having held that office in 1916-17. He also served as president of the U. S. Livestock Sanitary Association in 1910 and as secretary in 1908 and 1909. The latter date marked the last meeting of the Interstate Association of Livestock Sanitary Boards, at which time the name was changed to its present designation.

The Borden award of $1,000., medal and certificate, sponsored annually by the Borden Company Foundation and administered by the A.V.M.A., was awarded to Dr. Ralph B. Little of Princeton, N. J.

Doctor Little was cited for his outstanding research work which has contributed to the

control of diseases of dairy cattle. His original research on several major diseases of cows includes important studies having to do with the value of colostrum for newborn calves, immunity to brucellosis, udder infections, and mastitis studies. These and other problems

Dr. Ralph B. Little

have provided material for approximately 80 scientific articles which have appeared in current veterinary literature.

Doctor Little is a research associate at the School of Veterinary Medicine, University of Pennsylvania, and is also on the staff of the Rockefeller Institute.

Dr. Adam A. Husman

Dr. Adam A. Husman, of Raleigh, N. Car., was presented the national award of the A.V.M.A. for distinguished service to livestock health. Cited were his activities in five major disease eradication campaigns, including dourine, tick fever, bovine tuberculosis, brucellosis, and foot-and-mouth disease. Doctor Husman is the third winner of this award. He is past president of the National Association of Federal Veterinarians and has been a leader in A.V.M.A. activities, as well as those in state and regional veterinary societies.

The Humane Act award is made annually by the A.V.M.A. to the boy or girl who has, during the year, set an example by performing the most outstanding act of kindness to animals. This year the Awards Committee decided that Joe Brown, a 14-year-old boy of

Joe Brown with one of the horses saved from fire.

Nashotah, Wis., should be particularly commended for his action in the rescue of five valuable show horses from a burning barn near his home. The citation reads that when the boy saw flames shooting from the roof of the horse barn, he asked his sister to call the fire department while he rushed into the building and led out five horses, one by one. Only one of the five broke loose while being led out. After the rescue of the animals, the boy returned to the barn to pull out valuable show buggies and then assisted the firemen to extinguish the blaze.

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The record of losses among mink in four anthrax outbreaks on Wisconsin mink ranches points up the hazard to the fur industry due to this devastating disease. Source of infection in all was determined to be meat from infected animals.

PRINCIPLES OF ANIMAL HEALTH

R. R. DYKSTRA, D.V.M.
Manhattan, Kansas

South American Method for Treatment of Echinococcosis in Dogs

From a report issued by the World Health Organization, Geneva, we have learned that in some South American countries, notably in Patagonia, Argentina, and southern Brazil, there is practiced a system for mass treatment of dogs for echinococcosis. First, dog owners are notified to bring their dogs to some central area. The animals are to have had no food the previous evening. Then, an anthelmintic, consisting of a 1% aqueous solution of arecoline hydrobromide with sugar added, is administered orally, the dose being 10 ml. for large dogs which weigh more than 20 kg., and being progressively reduced for lighter weight dogs. The dog's weight is by estimate, as there is quite a margin of safety in the recommended dosage. The dogs are marked and, in order to control vomiting, the attendant is required to keep the dog's head elevated for ten minutes. Then the animal is allowed four hours of relaxation by being tied to a stake, followed by bathing or dipping in portable tubs containing 5% creolin solution. All excrement left by the dogs is buried in pits. During the four hour wait, owners are instructed about hydatosis by means of lectures, demonstrations and motion pictures.

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In regard to X-disease, or hyperkeratosis, of cattle, as briefed during a research conference held June 30-31, 1951 at the New York State Veterinary College, the following notes were made:

1. That "the free fatty acid fraction of the ether extract" of a processed animal feed concentrate produced symptoms of hyperkeratosis.

2. That a "particular batch of timothy hay from an identified meadow" when fed to a calf resulted, after five months, in the development of a "severe typical case of hyperkeratosis like the field cases."

3. A German wood preservative given in small diluted amounts through a rumen fistula tube caused "the hair coat to become rough and the skin thickened and slight hyperkeratosis developed." At another station, at least four calves developed symptoms of hyperkeratosis when the German wood preservative was experimentally used as follows: "direct contact of calf with wood painted with fresh material; feeding the German wood preservative; inhalation by calf (slight lesions); and indirect skin contact with the calf."

In our opinion, all of this splendid research work holds high promise of the ultimate solution of what has, during recent years, been a mystifying problem for many practitioners.

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A great deal of interest has been aroused in the medical profession by the wide publicity in the lay press of so-called new cures for tuberculosis. Actually, the new agents seem to be very promising though final judgment must be deferred until more experimental and practical evidence is available. The substance used is the hydrazine derivative of isonicotinic acid, known commercially as rimifon, and as nydrazid. This chemical, it appears, is bacteriocidal in that tuberculous guinea pigs treated with it, on autopsy, show no viable tuberculosis organism. The foregoing seems to distinguish the isonicotinic acid derivative from the results obtained in the handling of tuberculosis with some of the antibiotics, as viable tuberculosis organisms can usually be isolated in this latter line of treatment.

From reliable sources, it is learned that human tuberculosis patients treated by the oral route with this new agent started to gain weight in two or three weeks, and fever and malaise disappeared. In general, there has been no drug toxicity. Research indicates that the isonicotinic acid derivative is probably specific against the tuberculosis germ, as it shows no action *in vivo* against several other organisms. Though tuberculosis in dairy and breeding animals in the United States has, by slaughter and sanitation, been reduced to less than 0.5%, the new drug will interest veterinarians.

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Experience seems to indicate that in the spraying of cattle for the control of grub (*Hypoderma lineatum*) infestation in cattle, the best results are obtained when the sprayer shoots a solid stream of rather finely sprayed material from a cone jet. The solid stream results in much better hide penetration though it requires the use of more spray material. However, this is fully justified by the better effect of the solid jet.

PUBLIC HEALTH

JAMES H. STEELE, D.V.M., M.P.H.
Atlanta, Georgia

Rocky Mountain Spotted Fever

A recent report by R. R. Parker, *et al.*, points out that the cottontail rabbit contributes to the maintenance of his infection in nature. The isolation is reported on seven strains of Rocky Mountain spotted fever rickettsia from *Haemaphysalis leporis-palustris*, the common tick found in nature on cottontail rabbits in the Bitter Root Valley of western Montana. The strains isolated appear to be serologically and immunologically identical with a laboratory strain of Rocky Mountain spotted fever rickettsia. They differ from the laboratory strain in its low virulence for guinea pigs, although when injected into guinea pigs it affords protection against challenge with a virulent strain of spotted fever rickettsia.

The authors point out that the rabbit tick does not usually bite man, but indicate that it may be of importance in maintaining the rickettsiae in nature among rabbits. They observe that all stages of *Dermacentor andersoni*, the tick most commonly involved in the transmission of the infection to man in the western United States, is also found on rabbits. From the evidence at hand, it appears that this tick may be important in maintaining Rocky Mountain spotted fever in mammalian hosts which may infect *D. andersoni*, the common vector of the disease to man. The possibility also exists that man may become infected by handling rabbits infested with infected ticks.

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The Florida Board of Health has reported an outbreak of psittacosis in south Florida. Parrots, parakeets, and macaws have been found to be infected. The virus has been isolated from 11 different birds, including one parrot, and one macaw, in addition to nine parakeets. Two human cases in Minnesota have been traced to a parakeet purchased in a Florida aviary.

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Dr. Dean Fisher, Maine director of health, reported a milk-borne outbreak of salmonellosis in September 1951. The organism involved was *Salmonella paratyphi B.*, which was isolated from a young boy who was a nephew of the dairyman involved. Nine human cases were traced to this one human carrier who contaminated the raw milk supply.

Rabies in North Central States

A sizable outbreak of rabies was reported in 1951 in parts of Wisconsin, Minnesota, North Dakota, South Dakota, and Nebraska, which have previously been free of the disease. The skunk has been the most widely incriminated animal. In addition to skunks, cats, cattle, dogs, foxes, and mice have been reported to be infected. Fortunately, the problem has not spread to urban areas. In most instances, cities have taken the precaution to vaccinate and remove stray animals. The disease in mice has not been confirmed by animal inoculation.

A total of 103 rabid animals was reported in North Dakota up to December 15, 1951. Domestic animals included 37 cats, 14 dogs, three cattle, and one sheep. Wild animals included 19 skunks, 19 mice and rats, three minks, three raccoons, one fox, one squirrel. one muskrat, and one gopher.

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Tapeworms

An interesting report on the problems of certain parasitic diseases in Connecticut was recently published. Among the data are included the fish tapeworm, rat tapeworm, and beef tapeworm, which are shown in the accompanying table.

It is surprising to note that the incidence of beef tapeworm is much higher than fish or the common tapeworm in man, which is usually found in rats and mice.

From Parasitic Disease Data for the Years 1940-1950, Inclusive, Connecticut State Department of Health, Bureau of Laboratories

Year	No. of Specimens	*D. latum*	*H. nana*	*T. saginata*
1940	509	0	0	7
1941	5,625	0	0	2
1942	507	0	0	0
1943	709	1	0	2
1944	886	1	0	2
1945	883	0	0	7
1946	2,233	1	0	6
1947	2,557	0	0	10
1948	3,482	0	0	7
1949	3,377	0	1	6
1950	3,592	2	0	8

POULTRY PRACTICE PROBLEMS

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Frederick, Maryland

Fowl Pox

Fowl pox, a disease of the unfeathered parts of birds' bodies, is caused by a virus and is characterized by the formation of pustules on the skin and mucous membrane. It is most commonly seen in chickens, turkeys, pigeons and canaries but has been reported in sparrows, pheasants, quail, partridges, and guinea fowl. Reports have also been made of the disease in water fowl, but they are not well substantiated.

There are three recognized types of the virus as follows: chicken, pigeon and canary. There is also good evidence that there exists a strain especially adapted to turkeys. Immunity is conferred by an outbreak and immunization of chickens and turkey flocks by the use of a live virus vaccine is routine practice in areas where these birds are raised. Pigeons and canaries do not respond as well to artificial vaccination even with their own specific types of virus. Under normal conditions, the best vaccine to use for protection of chickens and turkeys is one prepared from a chicken pox virus. In laying flocks of chickens it may be advisable to use a pigeon pox vaccine, because the reaction is not as severe, but also the duration of immunity is short. If a flock of chickens that has been vaccinated with pigeon pox vaccine is to be kept for more than six months, it should be revaccinated during the molting period with virus of chicken origin.

Turkeys should never be vaccinated with pigeon pox virus. Even when virus of chicken or turkey origin is used on turkeys the duration of immunity is seldom longer than six or seven months. In many turkey-rearing areas, it is necessary to revaccinate the breeding flock after six or seven months. This is especially true in mild climates where mosquitoes are prevalent in January to March.

Mosquitoes have been proved to be transmitting agents of the disease to both chickens and turkeys. They may be the cause of the disease spreading from a recently vaccinated flock or from a recent outbreak to a pox-free flock in the same vicinity. Outbreaks in turkey poults from a few days to a few weeks of age have been observed where the only possible source was from mosquito-carried virus from recently vaccinated chicken flocks in the same area.

The purpose in this brief discussion is to remind readers that there is such a disease, which is likely to be forgotten because of the fact that in most well managed flocks it is kept under control by vaccination. Space will not permit greater detail, but it is suggested that you review the disease in any good textbook on poultry diseases. Here is a disease that does respond to vaccination and practitioners can render a valuable service to poultrymen by aiding them in a sound prevention program.

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Several years ago, Agricultural Extension Services throughout the United States put on a yearly campaign with the slogan Swat the Rooster as an important means of improving egg quality. The campaigns did a marvelous job in preventing fertile eggs from being produced for eating and cooking purposes. The slogan is still a good one and needs to be used in rural communities whenever farm flock owners get careless and leave males with laying flocks kept only for production of market eggs. Veterinarians doing a country practice should encourage farmers to eliminate the rooster from market egg producing flocks. Another service that can be given to poultrymen clients is information on culling flocks at frequent intervals to improve the flock health as well as its efficiency.

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An excellent reference, Recommended Nutrient Allowances for Poultry, designates a booklet available from the National Research Council, 2101 Constitution Ave., N. W., Washington 25, D. C. Similar booklets on the same subject are available for swine, beef and dairy cattle, sheep and horses, and can be obtained for 50 cents each. Each is a summary of a report of the National Research Council, Committee on Animal Nutrition and is prepared by experts in their respective fields.

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If diethylstilbestrol is used for the purpose of tenderizing and fattening turkeys and chickens, care should be taken to inject it correctly. It is usually administered in a pellet which should be injected beneath the loose skin of the neck just back of the bird's comb. If this method is followed, there is little danger of any residue being left in an edible portion of the bird.

VETERINARY PARASITOLOGY

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Phenothiazine for Gapeworm Infestation

The continuous feeding of 3 to 4% phenothiazine in the mash, according to work in England,[10] affords almost complete protection against gapeworms, *Syngamus trachea*. The measure is probably the only practical one so far devised for controlling these parasites in pheasants and partridges, which are not easily treated with barium antimonyl tartrate, but may also prove satisfactory for chickens and turkeys. Aside from the needed confirmation of the regimen, the only risk to be determined for poultry is that of possible interference with weight gains. The regimen, as will be noted, involves a higher concentration of phenothiazine than the 0.5% employed for the control of cecal worms and blackhead. Since young birds are the principal sufferers, a short period of medication is a hopeful prospect.

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British Question Our Position on DDT

Few, if any, informed persons now question the fact, and paramount significance of, chronic DDT toxicity. Nevertheless, there have been some honest differences of opinion regarding the practical hazards associated with intelligent use of the chemical. A recent British publication[11] notes the conclusion of American workers[12] that as little as 5 p.p.m. in the diet of rats can produce hepatic injury. It also notes that liver changes were not found in dogs, cats, monkeys, nor the larger experimental animals. Referring to the important bearing of these and other findings on the "permitted limit of DDT in foodstuff for human or animal consumption", the publication observes that, "The implication that a concentration of DDT might be harmful has been accepted in America (Dunbar, 1949)."

In the experience of Cameron and Cheng, the authors of the British article, rats that were individually dosed with 0.36, 3.6, and 36 mg. per kg. of body weight, six days weekly, showed no histological changes in liver cells when they were autopsied after periods of 33, 43, and 60 weeks of medication. Such changes as were noted are said to have been found to an equal extent in control rats, and none of the treated rats showed lesions that could be regarded as characteristic of chronic DDT poisoning. The doses used were calculated to be the equivalent of 3.5, 35, and 350 p.p.m. in the diet.

It can be predicted that this subject will be discussed further, since "In 1947, a series of long-term (DDT) feeding experiments on rats, fowls, sheep, pigs, and cattle were started by the Agricultural Research Council."

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Reports from Europe[1,2,3,4] suggest that benzene hexachloride is being used safely and effectively for the treatment of human pinworm infection. The product used and the regimen employed are not well defined, although there is reference to the use of 25 mg. tablets of gammexane and total doses of 225 to 600 mg. Several successfully treated patients had been refractory to other medicaments. Gammexane has also been used as an ascaricide for dogs[5].

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In cattle slaughtered in Edinburgh, the incidence of cysticercosis was 0.4% in 1948 and 1.16% in 1949, but a search did not establish an increased incidence of human infection with *Taenia saginata*. Interestingly, the infection rate was higher in women than men, suggesting that infection may be acquired in the kitchen where raw beef is available[6].

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Acanthosis Nigricans: A Case Report

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Acanthosis nigricans, also known as keratosis nigricans and papillary and pigmentary dystrophia, is a skin condition occasionally observed in dogs. It is characterized by bilaterally symmetrical hypertrophy of the papillae, increase of pigment in the stratum corneum,

Fig. 1. Acanthosis nigricans, lateral view.

and the malpighian layer of the epidermis, deposit of pigment in the papillae and around the blood vessels and glands, and moderate thickening of the horny layer, especially in the folds of the skin[3]. The areas usually involved are the axillae, popliteal spaces, scrotum, anal region, under-surface of the tail, abdomen, under surface of the neck, and the skin at the angle of the mouth, the eyelids and lips.

There develops a swelling and furrowing of the skin, and shedding of hair. Greyish-blue or blackish pigmentation appears and numerous papilliform swellings form. The skin is criss-crossed with ridges or folds and becomes harder and rougher than normal. There is very little exfoliation.

The exact cause of the disease is not known. Zepp[7] believes that the majority of cases are associated with infected anal glands and that the condition will clear up if anal glands are removed and the affected areas treated with 5% sulfathiazole ointment. Stark[6] does not give an opinion as to the possible cause of acanthosis nigricans but reports that he has effected permanent relief of symptoms in 14 to 20 days by the liberal application of colloidal sulfur lotion to the affected areas. Munson[4] expresses the opinion that it is probably the result of long-standing irritation. He has had good results by maintaining scrupulous cleanliness by frequent bathing with medicated soap, the application of colloidal sulfur lotion and the use of tonics designed to improve general condition. Whereas the absence of anal gland secretions on the surrounding skin may aid in clearing up this disease, he opines that it is doubtful if these secretions are sole responsible cause. Several cases were observed where removal of the anal glands failed to relieve symptoms[4].

Fig. 2. Acanthosis nigricans, close-up view showing furrowing of the skin and ridges.

On October 8, 1951, an eight-year-old male black Shepherd was brought to the Kansas State College Clinic with a "skin irritation" of six-months standing. No other information could be obtained. As illustrated (Fig. 1 and 2) the anal region, scrotum, sheath, ventral abdomen and chest, flanks, medial surface of the thigh, axillae and medial surfaces of the fore-arm were involved and was diagnosed as acanthosis nigricans.

The dog had a normal pulse and temperature, was friendly and in good spirits, and ex-

hibited no signs of discomfort or distress. Euthanasia was performed as requested.

The surface of a small portion of the affected area of the skin was thoroughly cleaned with alcoholic roccal® and a section of it was then cultured. A mixture of Staphylococcus and blunt Gram-negative rods were obtained. These are considered normal flora of the skin. A skin scraping and a section of the skin were stained for fungus by the Hotchkiss-McManus method. Fungus was not present.

Fig. 3 is a section of the skin showing the greatly increased stratum corneum, the crypt

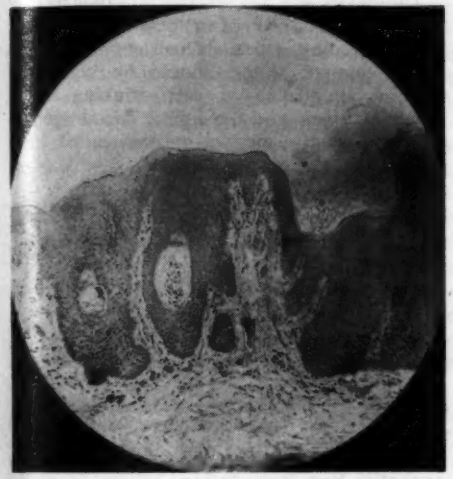

Fig. 3. Acanthosis nigricans, dog, microscopic section showing increased cornification, cell "nests", crypt of "furrow", and large papillae extending from the corium.

or "furrow", cornified cell "nests", and large papillae extending from the corium. Cells containing large amounts of dark pigment can also be seen.

Microscopic sections were made of the anal gland, prostate, testicle, adrenal, liver and kidney in an effort to determine if infection or dysfunction of any of these organs might be associated with acanthosis nigricans. The anal glands were small but otherwise normal. The prostate was normal. The germinal epithelium in the seminiferous tubules was about six to eight cells deep but the spermatogenesis apparently was not taking place. There were no sperm in the lumen or attached to the germinal cells. There was no sign of any inflammatory reaction. The adrenal gland appeared normal. The liver showed slight degenerative changes in some of its cells. There were some slight degenerative changes in the convoluted tubes of the kidney. However, these were not considered significant.

According to various authors,[1,2,5,7] the condition of the skin, in non-parasitic cases of dermatitis, is influenced by the following:

1. Hereditary or familial predisposition.
2. Contact irritants such as irritating antiseptics, soaps or oils.
3. Constitutional disturbances, gastrointestinal, endocrine, renal.
4. Allergy and hypersensitivity.
5. Nervousness (neurogenic factor).
6. Nutrition (improper diet).
7. Infectious diseases.
8. Extremes of heat and cold.
9. Bacterial infection (pyogenic factor).
10. Dry skin factor.
11. Oily skin factor.
12. Intestinal and blood parasites.

In the particular case with which this report is concerned, it is reasonable to conclude that the internal organs examined were not responsible. The anal glands were small and their secretions scanty. No constitutional disturbances, intestinal or blood parasites, infectious diseases or bacterial infections were detected. The dog had lived on a farm most of his life. It was not possible to obtain information concerning his hereditary background, the diet he received, peculiar habits, if any, or his favorite hang-out (i.e., hay-mow, feed room, hog pen, etc.)

It would appear logical to postulate that acanthosis nigricans is the result of either a low-grade allergy to an unknown external agent or a mild irritating substance acting on the skin over a long period. Good results obtained with colloidal sulfur lotion would tend to bear this out.

The writer wishes to express his appreciation to the Pathology Dept., Kansas State College, for the preparation of tissues and to Dr. W. M. McLeod for the photomicrograph.

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Cystic Calculus in the Horse

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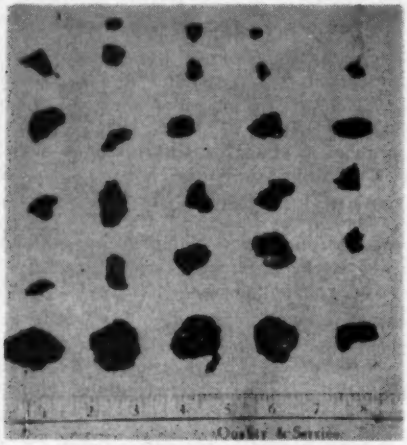
Bellevue, Nebraska

A six-year-old Tennessee Walking horse was presented with a provisional diagnosis of kidney stone. The owner had noticed that the horse had difficulty in urination, and dribbled blood-stained urine. These symptoms had occurred irregularly for six months. The animal had been treated for a kidney infection.

Diagnosis

Rectal examination revealed a calculus, about the size of a hen's egg, in the bladder. It appeared to be embedded in the mucosa.



Fragments shown above were taken from the case described and weighed 86.4 gm.

Treatment

Anesthesia was accomplished by epidural injection of 25 cc. of 1% solution of procaine hydrochloride. The surgical area was further anesthetized by injecting a 1% procaine solution along the line of incision. A rubber catheter was passed into the bladder.

Incision was made just below the anal sphincter, on the median line, which was extended downward immediately ventral to the ischial arch and deepened through the tissue until the urethra was exposed, recognized by the presence of the catheter. A longitudinal incision was made in the urethra, large enough to admit a pair of lithotomy forceps. One hand was passed into the rectum to help guide the instrument. The calculus was grasped with the forceps and worked loose from the mucous membrane in which it was embedded; then grasped and crushed. Larger pieces were extracted with the forceps, the smaller ones flushed out with warm water. The incision was allowed to heal as an open wound. Complete recovery was apparent after two weeks.

Discussion

The calculus was rough, crystalline, and brown in color. This type usually contains about 87% calcium carbonate, the remainder being magnesium carbonate, calcium oxalate, and organic matter.

The owner was advised that in order to prevent the formation of a new deposit, any fault in feeding, especially one combined with a privation of water, should be corrected; that an abundance of soft drinking water should be given, and the animal encouraged to drink. A laxative diet was recommended, having a high vitamin A content.

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Brucellosis in Cows

(*Continued from page 326*)

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Feline Infectious Enteritis

(*Continued from page 330*)

destroyed. Three of the four kittens which died showed normal blood counts the day before death. Autopsy revealed lesions similar to those seen in infectious feline enteritis. One kitten was apparently immune and survived.

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Scientific research finds facts and furnishes proofs.

ABSTRACTS OF LITERATURE

Effects of High Environmental Heat[10]

Effects of high temperatures on warm-blooded animals are considered commonly under three headings: (1) Heat cramps, which occur in animals that sweat, horses, when loss of fluid and salts by sweating is replaced by water alone; (2) heat exhaustion characterized by pronounced fatigue and body temperatures elevated by not more than one or two degrees; and (3) heatstroke or sunstroke of sudden onset. Extremely high body temperature in heatstroke often is recorded, attaining recording limits on fever thermometers.

Body temperature of mammals is maintained within normal range by the thermoregulatory centers in the central nervous system. These may be stimulated to initiate change in circulation by either changes in the temperature of blood flowing through them or reflexly in response to changes in skin temperature. The sum of these, according to the author, amounts to dilation of the cutaneous vessels with a corresponding constriction of internal vessels.

Heat cramps are not so common in animals, though in horses they may resemble colic. Heat exhaustion usually comes on slowly, while heatstroke is of sudden onset. The latter is of primary concern to veterinarians.

Premonitory signs such as staggering, visual disturbances, and difficulty in swallowing may precede collapse. Temperature is five or more degrees above normal. In the acute or "pink stage", mucous membrances are reddened, respiration is full and rapid, breathing is deep, and pupils usually dilated. On worsening, the "gray stage" is approached. In this stage, the skin becomes dry, pulse is rapid but weak, breathing shallow, and mucous membranes are pale to cyanotic. Circulatory collapse may follow. The author states that the total heat load and not direct sun rays on the head are responsible for heatstroke, contrary to popular conception.

Treatment of affected animals should be prompt and vigorous and directed to lowering body temperatures. Small animals may be dipped in cold water or placed under a cooling shower while providing for increased circulation of air. Spraying cool water over large animals and providing ample circulation are helpful in reducing temperatures. Intravenous injections should be avoided in the acute stages because of evident stress on the circulatory system during this period. After partial reduction in temperature, slow administration of cool hypertonic solutions of dextrose and saline is beneficial. Stimulants such as the long recommended camphorated oil are contraindicated according to results of recent pharmacological studies.

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Value of Sulfanilamide on Fertility of Bull Semen

A six-month trial to determine the value of sulfanilamide added to semen diluter was conducted at the Dartington Hall Cattle Breeding Center, Totnes, South Devon, England[11].

The collection from selected bulls was divided into two parts, each of which was diluted with egg yolk-citrate buffer. To buffer used for one part, 0.3 gm. of sulfanilamide was added per 100 cc. Inseminators were supplied equal quantities of each sample and were instructed to use them alternately. Results were judged by non-returns at three weeks and three months.

From data collected, no significant differences were apparent between conception rates of the two groups of cows or the incidence of early fetal death.

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Follicle Hormone Used for Castrating and Fattening Sows

A synthetic follicle hormone was used on 40 sows. It was found[4] that castration-like action was obtained with one dose of 85,000 international units per kilogram (2.2 lb.) bodyweight. Best suited for this procedure are sows weighing 200 to 265 lb. The exclusion of libido was accompanied by weight gains which averaged 1,085 gm. (almost 2.5 lb.) daily for 11½ weeks.

The meat of the slaughtered animals was fatter and of better quality than that of controls.—*R. S.*

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BOOK REVIEWS

Books reviewed in this department and any other books, if available, may be obtained by remitting the published price to the Book Department, Veterinary Medicine, Suite 803 Livestock Exchange Building, Kansas City 15, Missouri

ETIOLOGY, DIAGNOSIS AND CONTROL OF INFECTIOUS BOVINE MASTITIS, by *I. A. Merchant,* D.V.M., Ph.D., dean, and *R. A. Packer,* D.V.M., Ph.D., professor of veterinary hygiene, Division of Veterinary Medicine, Iowa State College, Ames. 85 pages. Burgess Publishing Co., Minneapolis 15, Minn., publishers.—Price $2.00.

This monograph briefly covers many aspects of a most important disease of dairy cows. Material is divided into five chapters and treats the predisposing factors; bacteriology, diagnosis, control, and treatment of mastitis. A reference list citing approximately 100 selected papers appearing in periodical literature is appended.

What is known and matters requiring further investigation are set down briefly to give a foundation for further study. The chapter on diagnosis will be most useful to practitioners as commonly employed, practical procedures are outlined and their comparative values pointed out.

In regard to treatment, the authors make the significant observation that there has been no drug yet discovered which will eliminate all types of infectious agents responsible for bovine mastitis. Even combinations or general use fail in many instances. In view of this observation, attention is focused on diagnosis as a key to successful treatment. Owners need to have this point emphasized to show the lack of justification of promiscuous use of one or another of various remedies offered.

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THE MODERN DOG ENCYCLOPEDIA, edited by *Henry P. Davis.* 626 pages, 528 plates. Stackpole and Heck, Inc., Harrisburg, Pa., publishers, 1952. — Price $10.00.

Authoritative estimates place the dog population in the United States at 20,000,000, which approximates one dog for every seven persons. From this figure alone, one can appreciate the interest in these animals for pets, for hunting, for guards or companions, or as working dogs for herding, police or war duty, etc.

In this book, of encyclopedic size and scope, the editor, a competent judge and breeder of sporting dogs, has solicited and included specialized material derived from many sources to supplement his work. Reference material of interest to dog owners, be they fanciers or simply owners of loved pets, is included. The book is comprehensive.

The first section is devoted to the origin of the dogs and early history, as man's ally and their use in sport. All breeds officially recognized by the American Kennel Club are given individual treatment which includes, in addition to origin, well known breeders and description and standards of the A.K.C. or specific breed club. Some other American breeds are also included; all illustrated by reproductions of photographs of dogs of bench show or field fame.

Breeding, care, feeding, housing, and other husbandry matters are well prepared. The author of a brief discussion of some 6½ pages entitled Some Common Dog Diseases, obviously is unacquainted with the subject and this section would best have been deleted. Chapters on Dogs and the Law, and Training and Handling are commendable. Appended is much information regarding rules and regulations pertaining to shows, field trials, records, etc.

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Toxicology in Canine Medicine

(*Continued from page 334*)

Clinical Toxicology, ed. 2, Philadelphia, Lea & Febiger, 1948.

31. Ward, Justus C., Rodent Control with 1080, ANTU, and other War Developed Toxic Agents. *Am. Jour. Pub. Hlth.,* 36:1427, 1946.

32. Ward, Justus C., Personal communication, 1951.

33. Wharton, Francis, and Stille, Moreton, Treatise on Medical Jurisprudence, ed. 2, Philadelphia, Kay & Brother, 1860.

34. Williams, R. T., Detoxication Mechanisms, New York, John Wiley & Sons, Inc., 1947.

35. Witthaus, R. A., and Becker, T. C., Medical Jurisprudence Forensic Medicine and Toxicology, New York, William Wood, 4 vol., 1894.

36. Wooley, Frank K., (Deputy Administrator, Production and Marketing Administration) Testimony House Committee Agriculture, Feb. 20, 1951. *Agr. Chem.,* 6:85, 1951.

37. Wormley, Theodore G., Micro-Chemistry of Poisons, ed. 2, Philadelphia, J. B. Lippincott Co., 1885.

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ITEMS OF VETERINARY INTEREST

It is estimated that meat output in the United States for 1952 will average approximately 4,200 lb. per farm.

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Cash sales of cattle, hogs, and sheep during 1951 set a new high record of $11,300,000.—*Report to National Livestock and Meat Board.*

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The addition of hyaluronidase to local anesthetic agents employed for infiltration anesthesia is gaining favor among veterinary surgeons.

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Vegetable crops supply a superabundance of energy for human nutrition needs, but are short in essential amino acids and vitamins. Foods of animal origin usually supply ample vitamins and proteins per unit of energy. Complementary merit of food elements derived from vegetable and animal origin is most important.

Brucellosis may cause sterility in man.—*S. N. L.*

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Summer training for veterinary students after their third year's work is completed has been offered by the BAI to encourage them to seek government employment at graduation.

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Forceful vomiting, referred to as projectile vomiting in human medicine, is characteristic of pyloric stenosis in infants and suggests a similar anomaly of dogs.

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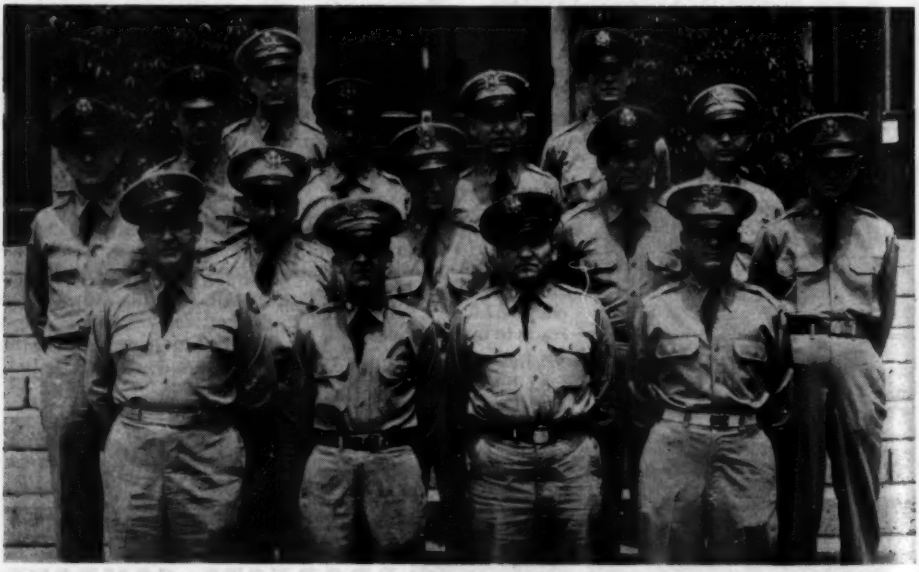
"In order to evaluate the present status of our mobilization program, it is necessary to go back to the start of the Korean war. . . . Our total military services numbered at that time less than 1,500,000. They have been expanded until today they total more than 3,700,000, and their effectiveness has increased in far greater proportion."—*Robert A. Lovett, Secretary of Defense.*



Fifteen Army Veterinary Corps officers last week completed the first advanced veterinary medicine course held at Walter Reed Army Medical Center's graduate school since 1939.

Designed to familiarize the students with the latest developments in veterinary medicine and basic medical sciences, the 17-week course included such varied subjects as the veterinary aspects of biological and chemical warfare, effects of nuclear energy on man, food, and animals, nutrition and deficiency diseases, and food control.

Front row (l-r): Lt. Col. Conley C. Isenberg, Lt. Col. Howard C. Maxey, Lt. Col. John Gale, Capt. William A. Bridenstine; second row (l-r): Major Burton C. Christopher, Major Charles B. Frank, Major Merida W. Castleberry, Major Seidel Stephens, Major William G. Sullivan; third row (l-r): Major Buford F. Bridges, Lt. Col. William G. Phifer, Capt. Lorenz L. Beuschel, Major William A. Nusser; fourth row (l-r): Major Elwin R. Prother, Major Omar G. Werntz.